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HEAD INJURIES AND THEIR MANAGEMENT

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PREFACE

In this monograph an attempt has been made to describe briefly the essentials in diagnosis and treatment of head injuries The objective has been to make this knowledge easily accessible and of practical value to residents as well as to the practicing surgeon and physician A representative bibliography has been added At least three excellent monographs on acute head trauma are already available one dealing with peacetime injuries and stressing their pathologic aspects (Evans, 1950) and the other two with war wounds of the skull and brain (Penfield 1941 Matson 1949) In addition many outstanding and much more comprehensive volumes on the subject of cranial and intracranial injury are at the disposal of the specialist (Munro 1938 Gross and Ehrlich 1940 Rowbotham 1949 Brock 1949) but these are mostly too extensive, and only too often unavailable, to be of immediate value to the doctor who must initially care for the patient and frequently decides upon subsequent management.

INTRODUCTION

In treating a patient with a head injury the doctor should decide whether he is dealing with a surgical or a nonsurgical (nonoperable) case, for all head injuries may be so divided.* To make such a decision one must have at least an acquaintance with the important surgical and nonsurgical lesions of the head and brain and the clinical picture they produce. Certain well-established, conservative measures should be used in the treatment of every head injury keeping in mind that all cases are potentially surgical. Fortunately, apart from cases with obvious externally visible head wounds and their complications, only a small per cent of serious injuries, in peace time, will require surgery for an intracranial hematoma or other less common lesion.

The purpose of this monograph is to state in simple terms what may happen to the head and brain following an injury and how such lesions and their complications may be recognized and managed.

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1

THE MECHANISMS OF HEAD AND BRAIN INJURY

HEAD INJURIES

A knowledge of some of the mechanical factors in volved in trauma to the head will aid one in under standing how various types of injury have very differ ent effects upon the skull and its contents[24, 37, 40, 88, 100]

An injury to the head may be either direct or indirect.

Direct Head Injury There are many ways in which the head can be injured. The movable head may be struck by a large moving object that jars the entire brain and skull or by a small object like a hammer that penetrates causing local damage without a gen eralized effect. A mobile head can be thrown against an immovable object, e.g., a sidewalk again produc ing an effect on the entire cranium and its contents

An immovable head braced against the ground or other firm substance may be generally crushed or burst by a large object or a relatively small area of skull and brain can be crushed, for instance by a small projecting portion of a jeep when the vehicle rolls over on the patient's head In this case as with a hammer wound there may be severe local brain

damage with no general effect on the brain and no loss of consciousness

Other direct injuries can result from penetrating instruments, bullets or shell fragments

Indirect Head Injury Injury to the brain or intracranial blood vessels at times follows the application of force to other parts of the body as when one falls in a standing or sitting position or on some other area of the body Brain injury may also occur when force is suddenly applied to the body by a rapidly moving object like an automobile or when, as a passenger in an automobile, one's head is thrown into violent motion on colliding with another car or on coming to a sudden stop

BRAIN INJURIES

The type and degree of injury is of importance, for upon this will depend to a considerable extent what happens to the brain and other intracranial structures (see Gurdjian, *et al*[37] and Rowbotham[88])

The brain may be injured by

1 Movement of the brain in relation to the skull
2 Distortion of the skull
3 Acute compression of the thorax
4 Penetrating wounds (open head injury)

1 Injury by Movement of the Brain in Relation to the Skull In closed head injuries the most important manner in which the brain is injured is by its movement in relation to the skull The movement may be linear, that is in a straight line, or rotatory

Brain Injury by Linear Movement If the head is thrown into rapid motion in a straight line, the

skull ceases its movement first and this often occurs abruptly especially if it comes into contact with a fixed object. The brain on the other hand for a short period continues its forward course and comes violently into contact with portions of the rigid skull or dural septa resulting in contusion or laceration At the opposite pole to that contused, the brain in moving forward apparently creates an area of dimin ished pressure and a suction injury to cerebral tissue or blood vessels may result At the same time vessels especially veins crossing the subdural space may be torn as the brain slides forward or backward in a straight line

Similar injury takes place at the onset of a rapid movement of the head in a straight line but in this case the skull is set in motion first and strikes the brain which has lagged behind in its motion Thus a contusion may occur at the site of the blow a contre coup injury in the opposite hemisphere and at the same time petechial hemorrhages in the brain stem (Fig 1)

The forces responsible for the above injuries will vary according to the rate of change of velocity with which the head is set in motion in a straight line—so-called linear acceleration—and to the abruptness with which motion ceases—deceleration (see under concussion)

Brain Injury by Rotation A blow to the head usually causes some rotatory movement of the skull. The motion of the skull is secondarily transmitted to the brain which may be contused or lacerated by bony projections or dural septa at the onset or ter mination of rotation. Holbourn believes that injuries due to rotatory movement are caused by shear strain [44]

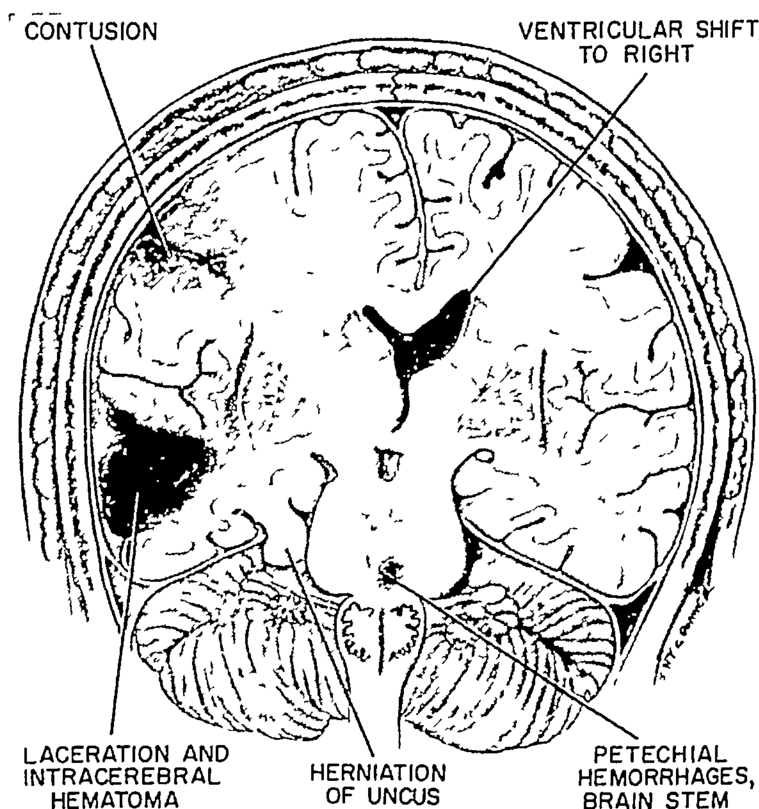


FIG 1 With the exception of the intracerebral hematoma the lesions shown are nonsurgical (nonoperable)

that results when one type of tissue slides across another, thus stretching or pulling apart the cellular structure Since the tissues of the brain itself are of different densities, shear strains also can take place within the brain or brain stem as a result of rotatory movement As with linear motion the injurious forces will vary with the rate of acceleration or deceleration

2 Injury of the Brain by Distortion of the Skull This type of injury is less frequent in civilian prac

tice than those already discussed. However certain traumas in producing general or local distortion of the skull may cause general or local crushing, pulping or bruising of the brain.

3 **Brain Injury from Compression of the Thorax.** Injuries of the brain especially in the form of petechial hemorrhages have been reported following blast or direct compression of the thorax.

4 **Injury of the Brain Due to Penetrating Objects or Missiles** This aspect of trauma will be discussed under surgical lesions of the skull and brain

2

NONSURGICAL CASES OF HEAD INJURY

In civilian practice over 97 per cent of all cases and about 90 per cent of severe cases of head injury suffer from a nonsurgical (nonoperable) craniocerebral injury if open head wounds are excluded * A high percentage of these nonsurgical cases have an injury of the brain reversible or otherwise but the treatment is conservative.

NONSURGICAL LESIONS OF THE BRAIN

Concussion. The fundamental basis of concussion is still unknown When the term concussion is used clinically it usually implies an immediate traumatic loss of consciousness lasting for a variable period and unaccompanied by evidence of structural damage to the brain There is a wide difference of opinion however as to whether prolonged unconsciousness can occur in the absence of demonstrable organic brain injury [2, 20, 44, 63, 69]

Denny Brown and Russell [20] define experimental concussion as the occurrence of an immediate traumatic paralysis of reflex function, which occurs in the

* See Chapter 3 for more detailed statistics.

cluded that concussion is the direct result of mechanical violence to cerebral cells

Another cause of concussion as demonstrated by Scott,[63] and Gurdjian and Webster [34] is that due to sudden brain compression This according to Denny Brown and Russell is much less common than that resulting from acceleration injury Compression concussion may occur with severe penetrating wounds certain depressed fractures and other types of injury that are accompanied by a sudden rise in intracranial pressure.

Walker *et al* [93] have provided evidence that experimental concussion is the result of intense excitation of the central nervous system at the moment of the blow to the head, which is manifested by a marked electrical discharge and a temporary breakdown of the polarized cell membranes of many neurones in the brain.

A review of many of the opinions held by investigators regarding the nature and cause of concussion may be found in the article by Denny Brown and Russell [19]

Contusion. A local contusion of the brain is an area of traumatic bruising in which there are varying degrees of swelling, perivascular hemorrhage disruption of cellular structure and even rupture of blood vessels The latter may give rise to an intracerebral hematoma (discussed under surgical lesions) (Fig 1)

Laceration. A laceration of the brain is an actual tearing of the neural and other tissues. There is no sharp dividing line between contusion and laceration and both lesions are commonly found for instance at the site of the blow at the area of contrecoup on the under surface of the frontal lobe, or at the tem

absence of visible lesions in the nervous system. They regard concussion as a generalized reversible "molecular reaction" induced by physical stress. Subconcussive blows, on the other hand, depress but do not abolish these reflex functions. They are of the opinion that pure concussion can account for a prolonged period of cerebral disturbance in man corresponding to the period of coma, semicoma and confusion which commonly follows a severe head injury. Such an injury they think may be associated with contusion and petechial hemorrhages in the brain stem, but these lesions are not responsible for the basic generalized cerebral disturbance of concussion.

Denny-Brown and Russell have shown that concussion can be produced when an animal's head is subjected to a sufficiently high rate of change of velocity. The change of velocity may be positive or negative and accordingly results in what they term either an acceleration or a deceleration concussion. Sudden acceleration sets up a complicated series of strains and distortions within the brain stem and cervical cord and it is these forces that are responsible for concussion as well as for hemorrhagic lesions when they occur. These hemorrhagic lesions are considered to occur immediately and are believed to be the result of direct injury and not due to prestasis, stasis and diapedesis.

Williams and Denny-Brown[108] have reported the electroencephalographic changes in experimental concussion. They found an immediate diminution of electrical activity from the whole cerebral hemispheres associated with concussion, followed by the appearance of slow waves. Reflex activity returned while the electrical activity was still diminished. They con

esses that cause a brief loss of consciousness and may result in no permanent injury On the other hand it may be that a prolongation or extension of this initially reversible process might lead to secondary changes of a permanent nature A third possibility is that more gross changes such as contusion or laceration are in some cases contributing to the unconscious state.

It is often very difficult to gauge the severity of the brain lesion on the basis of the clinical symptoms For instance a neurosurgeon can remove the frontal or right temporal lobe of the brain under local anesthesia and have the patient carry on an intelligent conversation throughout the procedure. Postoperatively the patient may be up the next day and be essentially symptom free. Similarly the greater portion of a frontal or temporal lobe is sometimes destroyed in an injury as with the jeep accident described and yet the patient remains conscious and alert. On the other hand a blow to the movable head may so jar it that a small hemorrhage occurs in the brain stem and the patient dies At the autopsy no other pathology may be found In fact trauma can cause death without producing any definite evidence of brain pathology at least of a gross nature

DIAGNOSIS OF NONSURGICAL CASES OF HEAD INJURY

All patients with a head injury should be under continuous observation and a description of the changing signs and symptoms recorded from the earliest possible moment Diagnosis rarely depends upon a single examination A detailed history is

poral pole where the brain has been forcefully thrown against the sphenoid ridge of the skull.

Brain swelling may occur locally around a contusion or laceration or be more generalized as a result of trauma. The swelling or edema in its milder form is presumably due to an increase in intracellular or extracellular fluid, but petechial hemorrhages in the cerebrum and brain stem and even actual liquefaction of involved areas can take place. As a result pathologic changes occur in the nerve and interstitial cells of the brain that may be permanent or transitory. The cause of the swelling may be the direct result of trauma to the tissues or secondary to injury of the vessel wall, which results in an increase in its permeability with escape of fluid, and possibly blood corpuscles (petechial hemorrhage), or even rupture of the vessel.[27,85,90]

Subarachnoid hemorrhage will occur when one of the vessels on the surface of the brain, or elsewhere is torn or ruptures and empties into the subarachnoid space. This will be true in laceration, frequently in contusion, and even as a result of severe edema. Lumbar puncture in such cases will reveal bloody spinal fluid.

Comment. Concussion may be brief or prolonged. It seems likely that concussion of short duration can occur without any permanent damage to the brain just as consciousness may be lost temporarily in an epileptic seizure due to a generalized physiologic disturbance.[97] Whether a loss of consciousness prolonged for hours or days can occur without permanent damage to the brain is more questionable. Unconsciousness of long duration might be due to a continuance of the same physical and chemical proc

esses that cause a brief loss of consciousness and may result in no permanent injury On the other hand it may be that a prolongation or extension of this initially reversible process might lead to secondary changes of a permanent nature A third possibility is that more gross changes, such as contusion or laceration are in some cases contributing to the unconscious state.

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DIAGNOSIS OF NONSURGICAL CASES OF HEAD INJURY

All patients with a head injury should be under continuous observation and a description of the changing signs and symptoms recorded from the earliest possible moment Diagnosis rarely depends upon a single examination. A detailed history is

essential and a complete examination, not only of the head but of the entire body, is required to rule out associated injury.

Signs and Symptoms and Their Pathologic Basis. There is one important difference between the signs and symptoms in the nonsurgical and the surgical case. The patient with a nonsurgical lesion rarely shows marked improvement followed by a worsening of his condition, although there may be some minor fluctuation in the severity of the symptoms. When a major fluctuation in the clinical course takes place in the manner mentioned, an expanding intracranial blood clot should be suspected.

The nonsurgical cases of head injury may be divided roughly into three groups depending upon whether the injury is slight, moderate or severe (see Symonds[97]).

Slight Degree of Injury. In the injury of slight degree the patient may be dazed for a few seconds or perhaps for half an hour, with or without loss of memory for the accident and for a period thereafter.

On the other hand the slightly injured patient may have a complete loss of consciousness for several minutes and may remain confused for a half hour or more. There is then an amnesia for the injury and rather frequently a so called retrograde amnesia for events immediately preceding it.

With slight injuries the neurologic examination reveals a loss of reflexes while the patient is unconscious. In some cases there are abnormalities in the neurologic examination for a variable time after the injury, indicating a mild, focal contusion of the brain and the electroencephalogram may confirm this. These findings are at times present in patients who

have been merely dazed and especially when a fairly prolonged period of amnesia has followed the accident

Some patients following a mild injury will complain of headaches dizziness and nervousness the cause of these symptoms is not clearly understood. They do not depend upon the severity of the trauma although the frequency of their appearance is greater in the more severe injuries (see under post traumatic syndrome)

Most cases of slight injury with loss of consciousness would be classed as examples of cerebral concussion with no permanent damage to the central nervous system This conclusion cannot be applied to all mild cases however and certainly not to those with evidence of focal cerebral contusion

Moderate Degree of Injury In this group there is loss of consciousness for perhaps five minutes to an hour or more The patient then goes through a stage of stupor during which he may be restless or violent followed by a period of drowsiness, confusion and disorientation The whole course lasts anywhere from a few hours to several weeks. Sometimes a patient who is improving will become more confused or difficult to arouse This will cause concern and is the type of thing that happens with an expanding blood clot. Such a patient will have to be watched with extreme care but usually the down hill course is of short duration and improvement sets in again or at least the patient s condition does not continue to become worse

On admission to the hospital these cases seldom show evidence of shock unless there are associated injuries or hemorrhage. There are usually some signs

of injury to the head such as contusion of the scalp, periorbital hematoma, discoloration of an ear drum from blood in the middle ear, or bluish discoloration of the scalp behind one ear There is frequently stiffness of the neck The temperature may be moderately elevated and later may rise to 102° or higher There is commonly incontinence A convulsive seizure or even status epilepticus is sometimes seen Lumbar puncture in a fairly high percentage of cases will reveal blood in the spinal fluid The spinal fluid pressure is moderately elevated in most cases but bears no definite relationship to the state of consciousness (see under subdural hematoma)

The neurologic examination will vary depending in part upon whether contusion or laceration of the brain is present There is arreflexia immediately after the accident, but later there may be some inequality in the deep and superficial reflexes on the two sides of the body The Babinski may be positive bilaterally or unilaterally Any motor weakness which is present was usually there from the time of the injury as a result of direct brain trauma If loss of motor power appears later then one must suspect an intracranial hematoma The pupils will vary in size and at times they are unequal A slight lower facial weakness is not uncommon Direct injury to one or more of the cranial nerves, of course, may occur

The prognosis must be guarded in this group so far as residual symptoms are concerned A good proportion will become completely asymptomatic within days to weeks Others will continue to complain for longer periods of headache, dizziness and inability to concentrate

The cause of the symptoms in these cases is a cerebral concussion combined usually with varying de

grees of brain contusion laceration edema and subarachnoid hemorrhage

Severe Degree of Injury The clinical course in this group is much the same as in the previous one except that the symptoms are more severe and the mortality high, especially in the first few days. The duration of unconsciousness is generally longer and about 20 per cent of patients although they may show some temporary improvement, continue in a stupor which becomes progressively deeper ending in death within about 48 hours.

Associated injuries of the face, long bones chest abdomen and elsewhere with their accompanying shock, are higher in this severely injured group and must be diagnosed and adequately treated

The patients who survive the injury for a few days will usually live if properly treated, but they frequently have a stormy course

Objective evidence of head trauma is nearly always present often with bleeding from the nose mouth or ears. Breathing is frequently difficult with accumulation of tracheal secretions cyanosis and signs of pulmonary edema Fluids cannot be taken by mouth sometimes for days or weeks. The patients are incontinent and may develop bed sores if not properly cared for Their temperature rises and in many will reach 106 or more unless treated assiduously This is especially true in the dehydrated patient or the alcoholic with delirium tremens. Epileptic seizures occur and cause hemiplegia from exhaustion of cortical cells and death if not controlled. At times the only manifestation of continuing seizures in an exhausted cerebral cortex will be a feeble twitching of one extremity or perhaps only of the fingers. When a hemiplegia occurs after a seizure it is often diffi

cult to know whether or not the paralysis is the result of a hematoma. All this is a clear indication of the importance of promptly controlling convulsive seizures when they appear.

The general neurologic examination will be essentially similar to that described for injuries of moderate degree. Stiff neck and bloody spinal fluid is the rule. Focal neurologic signs due to brain contusion and laceration will be more common. The effect upon the intellect, memory and disposition from generalized changes in the brain due to edema and petechial hemorrhage will be greater. Also in this group are seen those patients who remain unconscious in a more or less vegetative or decerebrate state for long periods as a result of hemorrhage or other damage to the brain stem.

The ultimate prognosis is more grave in these injuries of severe degree. There is a loss of memory for the accident and usually for a period preceding it. Post-traumatic amnesia or loss of memory for events following the injury may cover an interval of three months or more. Many will show intellectual impairment and emotional disturbance for a year or longer, but it is surprising how frequently patients will make an apparently complete recovery without disability. This lack of disability is more true of persons doing manual work. Individuals whose livelihood depends upon their intellectual capacity, however, may find themselves totally unable to carry on in their previous jobs. In the older age group even a minor head injury may be followed by marked intellectual impairment.

The patients who die as a result of their head injury as a rule show diffuse brain damage with con

tusion and laceration most commonly of the anterior temporal and inferior frontal regions In addition there is generally subarachnoid hemorrhage edema and scattered hemorrhages, commonly occurring in the brain stem.

CONSERVATIVE TREATMENT OF ACUTE HEAD INJURIES

There are a number of simple measures in the management of head injuries which if properly applied will appreciably lessen the mortality rate.

A patient is best transported from the scene of the accident in the supine position to avoid flexion of a possibly fractured spine, any fractures of long bones should be splinted hemorrhage controlled and ample blankets used if there are signs of shock.

On admission to the hospital a careful history is taken concerning the nature of the accident. Particular note is made of any loss of consciousness its duration and whether a lucid interval followed. Such a history may have to be obtained from relatives or witnesses of the accident.

Shock, if present is treated with transfusions and other measures regardless of the head injury Injuries elsewhere to the body whether facial spinal thoracic, intra abdominal to the extremities or elsewhere, must receive adequate care Cases of head injury uncomplicated by shock will stand transportation well and are best transferred to a hospital where neurosurgic treatment is available

Specific Measures in Conservative Treatment

1 A record of the blood pressure, pulse and respira

tion is made every half hour A complete neurologic examination is done at frequent intervals

2 The nurse is requested to test the state of con sciousness and motor power every half hour (especially in the first 24 hours) by having the conscious patient hold his arms in extended fashion In the semistupor-ous patient some evaluation of motor power can be obtained by painful stimulation or by raising each upper extremity and dropping it on the patient's face If power is present the arm will usually avoid striking the face

3 The unconscious or stuporous patient is placed on his side with a pillow under the head to avoid lat eral flexion of the neck and compression of the jugu lar vein In this position he avoids swallowing the tongue, vomitus or secretions He should be turned every two to three hours When a stuporous patient is on his back his breathing often appears of the Cheyne-Stokes variety because of obstruction by the tongue

4 Oxygen should be given to the unconscious pa tient by nasal catheter or in a tent A tracheotomy may become necessary to enable adequate oxygen ex change[25] Frequent aspirations for mucus are used as indicated

5 The temperature may rise to 106° or more in the seriously injured and must be controlled if the patient is to survive In such cases the temperature regulating mechanism in the hypothalamus is dis turbed[61] The patient's metabolism continues and heat is produced but cannot be lost unaided The temperature, therefore, should be recorded every half hour when elevated above 102° It must be kept below 102° with, if necessary, constant alcohol sponges,

multiple ice bags electric fans, etc. Usually a few hours of vigorous treatment will get the temperature down to where it can be controlled with alcohol sponges alone, but the nurse must remain constantly on the alert for a further rise

6 In all head injuries 1500 to 2000 cc. of fluid should be given every 24 hours by mouth or intravenously if necessary but it is probably best to delay intravenous fluids 24 hours in severe cases. The fluids should be nourishing if taken by mouth These cases do not tolerate dehydration any better than other sick patients. If the temperature is elevated or if the weather is hot another 1000 cc. of fluid are added. Patients with delirium tremens require about 4000 cc. a day

7 Careful electrolyte balance must be maintained.

8 Antibiotics are given routinely to the very sick as a prophylactic against meningitis from say a possible fracture into the paranasal sinuses or middle ear Such treatment is also preventive against hypostatic pneumonia which is prone to develop

9 Sodium phenobarbital is used for convulsive seizures, which are not infrequent following laceration or contusion of the brain The maintenance dose is gr i to gr ii hypodermically three times a day If the patient has repeated seizures (status epilepticus) it may be necessary to give up to gr viii of sodium phenobarbital in three or four hours If the seizures continue a full dose of Avertin by rectum can be used. Some employ intravenous sodium amobarbital or thiopental It is essential to get the seizures under control promptly in these sick individuals.

10 Sodium phenobarbital gr ii every four hours may be used if really necessary for restlessness If

this is not successful paraldehyde can be added It is well not to use morphine or Demerol Sedatives are best avoided when possible The patient should be allowed to move about the bed and be restrained only when essential

11 When a patient has been improving and then becomes drowsy caffeine sodium benzoate gr vii ss every four hours is useful to arouse him If the drowsiness continues or deepens then one must be on the alert for a surgical lesion

12 Should a suspicion of meningitis appear, lumbar puncture must be done immediately and the spinal fluid examined, cultured and the sensitivity of any organisms present tested with the various antibiotics For a meningitis give immediately sodium sulfadiazine Gm $\overline{\text{v}}$ intravenously in 500 to 1000 cc of fluid, and then Gm $\overline{\text{ii}}$ in four hours followed by Gm $\overline{\text{i}}$ every four hours thereafter for three to five days Large doses of antibiotics are also started immediately and later replaced by the antibiotic to which the organism is most sensitive

13 Lumbar puncture is useful in diagnosis It should be avoided during the first 24 to 48 hours, especially if the patient resists, as straining will raise the intracranial pressure and also the blood pressure and hence may cause intracranial bleeding After the first 48 hours lumbar puncture is indicated to determine the spinal fluid pressure when an expanding intracranial lesion is suspected Severe headache may be relieved by reducing the pressure by half Otherwise this method has not appeared of much value in treatment although there are opinions to the contrary

14 Drainage of blood and spinal fluid from the ear (otorrhea) is treated with antibiotics and by gently

sponging the outer ear with alcohol and covering it with a dry sterile dressing The cerebrospinal fluid and blood will be absorbed into the dressing Otherwise it would run into the outer ear become contaminated and then possibly drain back into the auditory canal and contaminate the cerebrospinal fluid. An otoscope should not be inserted into the ear canal when there is drainage of spinal fluid, but a large piece of wax or encrusted blood may have to be removed with care The drainage will usually cease in about one week with the above treatment

15 Drainage of cerebrospinal fluid from the nose (rhinorrhea) may occur when there is a fracture into one of the paranasal sinuses plus a tear in the adjacent dura and arachnoid The treatment is conservative for about four weeks unless complications arise. The patient is instructed not to blow his nose under any circumstances as he might blow air and bacteria into the intracranial cavity Antibiotics and sulfa drugs are given until the drainage ceases Surgery may become necessary (see under surgical treatment)

16 An indwelling catheter and tidal drainage is used for the patient who remains in prolonged coma and is not incontinent. Otherwise catheterization is done when indicated.

17 General care is given to the skin and pressure points to avoid bed sores

18 Roentgen ray pictures of the skull are not taken as an emergency and are contraindicated while the patient is seriously ill unless there are signs of an epidural hemorrhage or of a compound fracture. Frequently the patient is well enough to have roentgenograms in 24 to 48 hours after admission to the hospital

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SURGICAL CASES OF HEAD INJURY INTRACRANIAL HEMATOMAS

All cases of head injury whether "open" or "closed" are potentially surgical cases. They should be con tinuously under expert observation. Until operation becomes necessary the patient is treated like all other individuals with a head injury by simple common sense and conservative measures These conservative measures already have been described in the chapter on the treatment of nonsurgical cases

In this monograph surgical cases are divided into three main groups Intracranial Hematomas Surgical Lesions of the Scalp Skull and Brain (Chapter 4) and Other Complications Following Head Injury (Chapter 5)

The problem of deciding whether a case of acute head injury is surgical or nonsurgical (nonoperable) is an important one There are three pertinent points a knowledge of which will simplify the problem of diagnosis.

1 Surgical lesions of the scalp skull and brain in adults as defined in the following pages, can nearly always be recognized with ease by careful examina

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1 Surgical lesions of the scalp skull and brain in adults, as defined in the following pages can nearly always be recognized with ease by careful examina

tion of the head in the great majority there is an open wound of the scalp A rather uncommon exception is a simple, depressed fracture, which can be revealed by roentgenogram of the skull

2 Intracranial hematomas make up the great bulk of the other surgical lesions that must be diagnosed to save life and they are comparatively uncommon Statistics vary on the incidence of intracranial hematoma depending upon whether all degrees of head injury are included in the study or only severe cases that might be referred to a neurosurgical center The incidence is much higher among those with severe head trauma

Subdural hematomas constitute the largest group of intracranial hematomas and they have occurred in only about 1 per cent of all head injuries in some series,[24, 58] and in the neighborhood of 5 [24] to 10 [69] per cent of the severe cases in other series The other intracranial hematomas are the epidural and intracerebral hemorrhages Their incidence has been reported as much less than 1 per cent of all head injuries and as about 2 per cent of the severely injured by some observers [24] and as considerably higher by others (see below)

3 There are a few additional surgical complications following head injury, but apart from infection and arteriovenous aneurysm, surgery in their treatment is usually elective

In simple terms then it is estimated that if open wounds of the head are excluded, only about 2 to 3 per cent of all patients with a head injury, or about 8 to 10 per cent of the severly injured, develop an intracranial hematoma or other less common lesion that requires surgical intervention

Intracranial hematomas are either extradural subdural or intracerebral and may occur following any head injury whether this be of the "open" or closed variety

EPIDURAL HEMORRHAGE

An epidural hemorrhage is nearly always due to a tear of the middle meningeal artery either at the foramen spinosum in the middle fossa where it enters the skull or anywhere along its many branches (Fig 2A) The artery runs in grooves on the inner table of the skull and a fracture which crosses the vessel can easily tear it (Fig 2B) Usually a fracture must be present but, in rare cases, such a hemorrhage can occur without a fracture. When the bleeding takes place it strips the dura away from the skull and in so doing tears many other little branches of the artery and even may cause hemorrhage from pacchionian granulations near the longitudinal sinus. The larger the branch of the artery initially torn the more rapid and extensive the hemorrhage and the more severe and rapid the compression of the underlying brain and the development of clinical signs Occasionally an epidural hemorrhage may be due to a tear in one of the large venous sinuses or may occur in the posterior fossa.[20]

The incidence of epidural hemorrhage was less than 0 25 per cent in 30 000 head injuries of all types and about 1 0 per cent in 6000 severe cases studied by Echlin *et al* [24] but has been reported as 3 per cent by Munro and by Woodhall *et al* [2]

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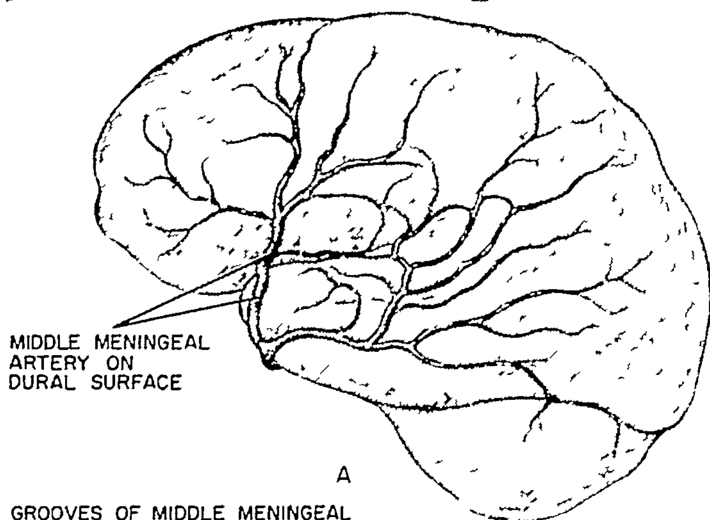
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hard object like a policeman's club or falls hitting his head on the pavement. He may be unconscious for a few minutes but gets up and walks along. *There is a lucid interval*. In a matter of minutes to an hour or more he develops a headache usually on one side of the head and may vomit. A convulsive seizure sometimes occurs. The patient then becomes drowsy and often confused. This is followed by a progressive hemiparesis to hemiplegia on the side opposite the hematoma, the arm frequently being involved first. The pupil on the side of the lesion often becomes dilated. In a rapidly developing hemorrhage from a tear in the main trunk of the vessel deep coma and decerebrate rigidity may take place within a few hours and operation is then frequently too late to save life.

There are variations in the syndrome. In many cases the course is not so rapid. The hemorrhage is less and may actually stop. A hemiparesis can be delayed for many hours and be relatively mild and in some instances is on the same side as the epidural clot. The motor weakness may only be recognized by the doctor on the day following the injury and the diagnosis not suspected until the history of a lucid interval is obtained. In fact an occasional case may remain drowsy with weakness of one side for a week or more before the diagnosis is made or a sudden worsening of the clinical condition takes place.

Cases with the history described in whom a *lucid interval* has occurred should all be suspected of having an epidural hemorrhage and explored as emergencies. Unfortunately some patients following a head injury remain unconscious and during this interval the epidural hematoma develops and keeps the



B

FIG 2 A The location of the middle meningeal artery explains why a hemorrhage from this vessel is on the outer surface of the dura B It is evident why a fracture of the skull may tear a branch of the middle meningeal artery as it runs in a groove on the inner table of the skull

dle fossa anterior to the ear it is usual to place the first burr hole about one inch anterior to the ear and a little above it. The head should be draped so that the incision can be extended down to the zygoma and upward even to the mid line Also it should be possible to extend it backward in horseshoe fashion to the mastoid process so that a skin and muscle flap can be turned and a large craniectomy rapidly carried out when needed.

When through a burr hole an epidural clot is exposed it should not be disturbed until the subtemporal decompression is enlarged to at least the size of a silver dollar The enlargement should be as much as possible toward the center of the clot If the clot is disturbed before adequate exposure has been made active bleeding, which is often absent at this stage may be set up and cause serious damage before it can be controlled. On the other hand, after a sizeable craniectomy has been carried out which can be done in a few minutes, the clot can be rapidly attacked with the sucker and spoon Bleeding may now occur from a large branch of the middle meningeal artery or even the main trunk at the foramen spinosum The surgeon can approach it immediately and cover it firmly with a piece of brain cotton or his finger until the field is cleared. Then the vessel can be coagulated clipped or tied and in addition the foramen spinosum may be plugged with bone wax and brain cotton if it is exposed Fortunately nature exposes the main bleeding point by having already stripped the dura away from the skull Hence when the artery is torn for instance low in the middle fossa no stripping of the dura is required to bring the bleeder into view At times there is additional bleeding from many tiny

patient in coma. These cases are usually not diagnosed. One should, however, be on the alert for them and explore a patient who shows deepening coma and develops a progressive hemiplegia with a dilated pupil on the opposite side. Nothing will be lost by operation in such a case and at times something may be gained.

The description of the rapidly developing signs and symptoms in epidural hemorrhage should make it clear why head injury patients must be observed so closely, especially in the first 24 hours after trauma. As pointed out under conservative treatment the frequent testing of the state of consciousness and motor power is most important if these cases are to be diagnosed. For an excellent description of epidural hemorrhage see McKenzie[69] and Munro and Maltby[70].

Surgical Treatment of Epidural Hemorrhage. In exploring for an epidural hemorrhage the same operative approach, positioning of the patient and method of anesthesia may be used as described in some detail under surgical treatment of subdural hematoma.

Before operating it is well to know if a fracture of the skull is present as it may give a clue to the point of tear in the middle meningeal vessel. Sometimes the fracture line will pass entirely posterior to the ear and then it usually will be the posterior branch of the artery that is torn. In such a case, a burr hole, in the usual position anterior to the ear, may reveal no evidence of a huge epidural clot lying entirely over the posterior aspect of the occipitotemporoparietal region. Occasionally the hematoma may be located in the frontal region.

When the fracture line traverses the side of the head from back to front or passes downward into the mid

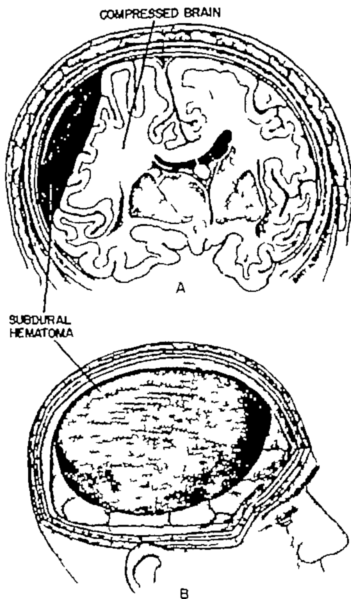


Fig 3 This is a common location of a subdural hematoma, but it frequently extends lower in the temporal region.

vessels on the surface of the dura and these may be tedious to control. Serious venous hemorrhage may also take place from the pacchionian granulations near the longitudinal sinus as mentioned. Occasionally this bleeding can be controlled only by gauze packing with perhaps, in addition, suture of the dura to the periosteum through burr holes placed over the convexities of the hemisphere. It may even be necessary in a very acute case to leave this packing in for a day or longer.

SUBDURAL HEMATOMA: ACUTE, SUBACUTE AND CHRONIC

A traumatic subdural hematoma or hemorrhage is the result of bleeding into the space between the brain and the dura mater from a vessel situated on the surface of the brain, or running between the brain and the dura (Fig. 3). The clinical course following a subdural hemorrhage may be acute, subacute or chronic.

Early reports of subdural hematoma were concerned chiefly with the chronic form as this was the type seen in neurosurgical centers. Wilfred Trotter in 1914 gave a classic description of chronic subdural hematoma and emphasized the traumatic origin of the lesion. In 1925 Putnam and Cushing described the pathologic features in detail and alerted the neurosurgical world to the condition. Since then many papers[11,15,22,24,28,32,35,43,57,58,68,69,84,102] have established chronic subdural hematoma as an important neurosurgical entity.

Descriptions of large series of acute subdural hematomas began to appear with the advent of emergency

the basis of the findings reported by Echlin *et al* [44] of 300 cases that the following observations on subdural hematoma have been made. Less than 5 per cent of 6000 patients with severe head injury were found at operation to have a subdural hematoma which is a lower incidence than that of Munro [69] who found 10 per cent in a more selected group

Etiologic and Pathologic Considerations in Subdural Hematoma. The most common cause of an acute or subacute subdural hematoma is a laceration of the surface of the brain with resultant hemorrhage into the subdural space over the surface of the cerebral hemisphere or very rarely over the surface of the cerebellum When the injury is mild which is commonly true in chronic hematomas, the source of the bleeding is probably rupture of one of the veins that bridge the subdural space Occasionally massive arterial hemorrhage may occur beneath the dura when branches of the middle cerebral artery are torn or a lacerated middle meningeal artery bleeds through a tear in the dura.

The formation of a collection of blood in the subdural space usually gives rise to few early symptoms unless the hematoma forms rapidly as in the uncommon case of active arterial or massive venous hemorrhage. The bleeding generally stops soon after injury and evidence of continued subdural bleeding rarely is seen at operation on subacute or chronic cases. Delayed symptoms in subacute cases it is believed therefore, rarely are due to slow hemorrhage The fatal course of many patients with acute subdural hematomas is as stated, generally the result of associated brain injury and its complications and not of the hematoma

Because the brain can adjust rapidly to a large sub-

TABLE 1 *Operative Experience with 300 Cases of Subdural Hematoma*[24]

	Type of Subdural Hematoma				
	Acute		Subacute	Chronic	
Time from injury to operation	2 to 48 hours	3 to 7 days	7 to 22 days	22 to 30 days	over 30 days
Total number of cases	50	43	132	20	55
Lived	5	21	101	18	38
Died	45	22	31	2	17*
Mortality	90%	51 1%	23 4%	10%	30 9%

* 12 of these 17 patients were in coma at time of operation Over all mortality 39 per cent

(From Echlin F A Sordillo S V R, and Garvey, Thomas Q, Jr Subdural hematona acute, subacute and chronic, J A M A In press)

neurosurgical services in big city hospitals[23, 24, 35, 55, 58, 68 69 102] With time it became customary to describe all subdural hematomas as acute or chronic

Later it was suggested that subdural hematomas be classed as acute, subacute and chronic A classification which includes only acute and chronic groups is misleading and fails to alert the profession to the fact that almost 50 per cent of subdural hematomas on a large emergency service are really *subacute* and require operation as a lifesaving procedure on the seventh to the twenty first day after injury[23, 24] (See Table 1)

Incidence of Subdural Hematoma In a series of 30,000 head injuries reported by Echlin *et al*[24] there were 300 operated cases of subdural hematoma, an incidence of 1 per cent, which is in keeping with the findings of Laudig, Browder and Watson[58] It is on

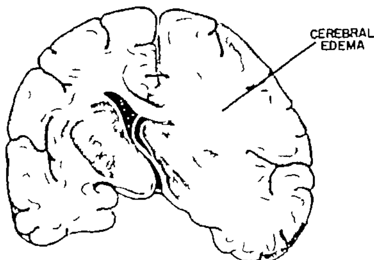


FIG. 4 Cerebral edema due to compression from a subdural hematoma

uncus and this can lead to infarction in the occipital lobe At the same time the third cranial nerve commonly is subjected to pressure by the uncus with resulting dilatation of the pupil and less often a ptosis.[44, 87]

Acute Subdural Hematoma. In this group are included all patients with a subdural hematoma who appear to require surgery as a lifesaving procedure during the first week after injury The majority of these individuals have sustained a severe head trauma resulting in fresh blood or blood clot in the subdural space and a seriously injured brain Because of the severe brain injury the mortality in the acute group is high during the first week and extremely high in the first 48 hours after injury even though the hematoma be removed

dural clot the patient may become asymptomatic or relatively so as he recovers from his direct brain trauma Most hematomas then liquefy, if they are not already in such a state, and become larger as they acquire more fluid, presumably through the process of osmosis, as demonstrated by Gardner[28] and others[29–31, 114] It is this increase in size of the hematoma over days to months with its resulting effect on the underlying brain, that causes progressive symptoms and death in the great majority of cases unless operation is carried out

In the chronic state the hematoma is most often liquid but may contain soft clot and usually is surrounded by a membrane at any time after the seventh day of its formation Occasionally the clot becomes organized to the consistency of liver or rarely is calcified

Pathologic Changes in the Brain[27–30] When a subdural (or epidural) hematoma expands it compresses the underlying cerebral hemisphere and displaces it medially As a result there can occur a serious disturbance in cerebral circulation and even some obstruction of the flow of cerebrospinal fluid from the lateral ventricles Interference with the venous outflow from the compressed hemisphere may lead to marked edema of the brain (Fig 4) and herniation of the medial portion of the temporal lobe (uncus) over the free edge of the tentorium[44–51, 56] and herniation of the cerebellar tonsils into the foramen magnum Pressure from the herniated uncus may interfere with the venous circulation of the brain stem and produce the equivalent of a decerebration through edema or actual hemorrhage into the stem[27, 55] The posterior cerebral artery at times becomes compressed by the

a head injury usually consist of currant jelly or later milk chocolate clots with varying amounts of fresh or old fluid blood and cerebrospinal fluid.

Of 43 patients in this category operated on the third to the seventh day by Echlin *et al* 22 died in most cases presumably as a result of their brain injury The mortality was 51 1 per cent which is much less than in those operated on within 48 hours following trauma The brief duration of the illness justifies placing these cases in the group of acute hematomas.

Subacute Subdural Hematoma Between the two extremes of the condition described as acute or chronic falls the group of subacute subdural hematomas. In this group which in one series[24] included almost 50 per cent of all subdural hematomas the head injury is most often severe There is usually blood in the spinal fluid and frequently a fractured skull Recovery from the brain injury occurs in the majority but is commonly incomplete and is followed in about one to three weeks by progressive signs of brain compression from the hematoma.

Most patients regain consciousness and about 25 per cent become mentally clear whereas the others continue to have some degree of drowsiness and disorientation Many of the more alert patients complain of headache Before operation approximately 80 per cent show progressive signs of brain compression evidenced by increasing drowsiness, usually fluctuating in nature and ending commonly in semistupor stupor or coma. In most instances there are abnormalities in the neurologic examination (see later under diagnosis)

About 90 per cent of the more alert cases can be safely diagnosed with air studies or angiograms before

Early Surgery Patients with a subdural hematoma in whom surgery has seemed indicated in the first 48 hours after trauma are usually unconscious for varying periods and then show slight improvement to the state where they are stuporous They rarely have a lucid interval Most of them then become increasingly comatose and often develop "localizing" signs such as a dilated pupil on one side and a progressive weakness of the opposite arm and leg

Occasionally the clinical picture in the first 48 hours will be much the same as described for an epidural hemorrhage if the bleeding is severe and the direct brain injury slight Such cases, if they have a lucid interval, are emergencies and must be operated promptly

In the series reported by Echlin *et al* over 90 per cent of 50 patients operated on in the first 48 hours after injury died, despite adequate removal of a large hematoma Death in this group was attributed largely, if not entirely, to direct brain injury, the hematoma being incidental Survival in five cases was due to removal of the blood clot, the injury being less severe

Later Surgery Patients with an acute subdural hematoma who present signs indicating operation on the third to the seventh day following injury usually regain consciousness but only occasionally are alert Most of them remain drowsy and disoriented and then become progressively drowsy or stuporous The onset of stupor may be sudden and not infrequently is precipitated by a convulsive seizure Neurologic signs of brain pathology are usually present and often indicate a "focal" lesion

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stupor appears In the remaining 80 per cent exploratory surgery becomes essential to save life at any time from one to three weeks after injury and carries a very good prognosis in the majority of cases, providing the patient has not lapsed into coma before operation

Pathologically the subacute hematoma consists of currant jelly or milk chocolate clots with different quantities of reddish brown to black fluid Occasionally the fluid is yellow or the hematoma may be organized Membranes may surround the clot at any time after the seventh day of its formation

In the group of 132 subacute cases reported by Echlin *et al*, the mortality was 23 4 per cent which is considerably lower than in the acute variety Unlike many of those with acute lesions, recovery from the brain injury had taken place or was occurring when the down-hill course was precipitated by compression from the subdural hematoma Improvement clinically and from the brain injury was usually less complete and less prolonged than seen in the chronic group Pathologically the hematoma as regards its stage of liquefaction or membrane formation, occupied in most instances a position between the acute and chronic types On the basis of time elapsed since the injury, clinical course, nature of the brain injury, and pathology of the hematoma, most of these cases are neither acute nor chronic and are well described as subacute [23]

Chronic Subdural Hematoma Patients with a chronic subdural hematoma usually have recovered from their head injury, which may have been severe but more often was mild, or there may be no history of trauma Several weeks to months later serious symptoms appear, often presenting a diagnostic prob

lem which may suggest brain tumor diffuse degenerative brain pathology or psychosis The history in the great majority of patients is one of mental deterioration for many weeks with gradual disorientation and confusion leading commonly to fluctuating drowsiness. Headaches rather commonly precede the appearance of mental symptoms Neurologic signs of organic brain disease often suggesting a focal lesion usually are present but they may be slight or absent. The diagnosis is not infrequently made during the course of a ventriculogram for a possible brain tumor or other expanding lesion Surgical evacuation of the hematoma, which as a rule is encapsulated, is curative in the majority of cases if the patient is not in coma at the time of operation

Patients operated on from the twenty first to the thirty first day after injury may present a clinical picture very similar to that described for the subacute cases and perhaps are better included with this group After the thirty first day the clinical course of the chronic subdural hematoma corresponds well to that just described.

Diagnosis of Subdural Hematoma. *If after a head injury a patient regains consciousness and is relatively alert (has a lucid interval) and subsequently in hours to weeks becomes progressively drowsy or stuporous it is essential that an intracranial hematoma be ruled out*

Such a clinical course developing in the first 24 hours is usually due to an epidural hemorrhage from the middle meningeal artery and as a rule is accompanied by a hemiplegia and a dilated pupil but occasionally a massive subdural or an intracerebral hematoma will give a similar picture These cases may be

saved by emergency operation *The vast majority the other patients operated in the first 48 hours, w have remained stuporous from the time of the ac dent, are suffering from severe brain injury and w not be benefited by surgery even though localizi signs are present and a subdural hematoma is moved* A rare exception is an epidural or large su dural hematoma unaccompanied by fatal brain traur that develops while the patient remains stuporous a is usually impossible to diagnose

After the first 48 hours have elapsed since the jury the most common and valuable symptom of subdural hematoma is progressive drowsiness, oft with disorientation and generally fluctuating in ch acter Headache may or may not be present In t chronic lesion headache and a gradual mental c terioration frequently precedes the drowsiness T clinical picture of an intracerebral hematoma or tum faction from local, traumatic softening[7] may close simulate that produced by subdural hematoma

Abnormal neurologic signs, although they may minimal or absent, are usually present and often gra ually progressive These include papilledema whi is not common during the first two weeks followi injury Frequent findings are an inequality of t pupils, less often a dilated pupil, a slight lower faci weakness, unequal deep reflexes, unequal abdomina and a positive, often bilateral Babinski Progressi hemiparesis even to hemiplegia as a result of a su dural hematoma occurred in 43 per cent of acute cas in one series,[24] 20 per cent of subacute and 42 per ce of the chronic A slight drift of one of the ou stretched arms is a frequent and very valuable sig

especially when it is increasing in severity A con vulsive seizure in any of these cases may precipitate a hemiplegia. Other less frequent signs include aphasia, homonymous hemianopsia a third or sixth nerve paralysis, a hemisensory loss, and in chronic cases catatoniclike posture tremor and unsteady or shuffling gait.

Some patients with a subacute or chronic subdural clot do not show progressive drowsiness or mental change, at least at first but may complain of severe headache with or without neurologic signs. Papil ledema or an elevated spinal fluid pressure may be the only positive diagnostic signs that this group pre sents Headache of itself is of course by no means indicative of an intracranial hematoma Actually drowsy patients usually do not mention headache

The diagnosis of subdural hematoma is not gen erally made on a single examination but on the pa tient s clinical course It is the increasing severity of the signs and symptoms that are important since those present from the time of injury are usually due to laceration or contusion of the brain

In summary it may be safely stated that if, after a head injury a conscious patient at any time from hours to weeks or even months *begins to show a progressive down hill course* in mental status or in neurologic signs indicative of an intracranial lesion he must be regarded as having a subdural or other intracranial hematoma until it is ruled out

Nonclinical Aids Although the clinical picture provides the most valuable criteria in the diagnosis of subdural hematoma there are certain nonclinical examinations that may be of great help These in

clude a shift of the calcified pineal body in the skull roentgenogram, pneumo encephalography, ventriculography, electroencephalography and angiography

Air studies or angiography are not well tolerated by the very sick patient with a subdural hematoma and bilateral temporal (and/or other) burr holes are safer and nearly always diagnostic

Electro encephalography has not been of much help in acute cases but in the subacute and chronic ones has been of great assistance especially when repeated EEGs are done and reveal a progressive abnormality (see under electro encephalography)

Localizing Signs Abnormal neurologic signs due to subdural hematoma although indicative of an organic lesion are frequently not of "localizing" value[24]

Localizing signs of some degree occur in nearly 50 per cent of the cases, but correctly indicate the side of the hematoma in only about half this number In the other patients with localizing signs these are either falsely localizing or there is a bilateral hematoma

An actual hemiparesis is present in almost a third of the cases, but in about one quarter of these is on the same side as the hematoma

An inequality of the pupils will be found in about half of all cases A slight to moderate enlargement of one pupil is of no localizing significance A marked dilatation of one pupil occurs in about 20 per cent and when present is correctly localizing in approximately 60 per cent In the others it is either opposite to the hematoma or there is a bilateral lesion

Convulsive Seizures A convulsive seizure occurring before operation in patients with a subdural hematoma is surprisingly common, being recorded in as high as 20 per cent of patients in one series[24] Pre

sumably a lacerated or contused brain is the cause or a contributing factor This high frequency of seizures in the presence of brain injury and subdural hematoma is a serious complicating factor and frequently precipitates a hemiparesis or a continuing stupor in an already drowsy patient.

Pulse Cerebrospinal Fluid Pressure and Blood Pressure Although Browder and Meyers have shown that there is no constant relationship between pulse rate, blood pressure CSF pressure and state of consciousness in cases of subdural hematoma there are certain observations that deserve comment. A slow pulse is found in at least one third of the cases and an elevated CSF pressure in about two-thirds A rise in blood pressure occurs in approximately one third and in about 50 per cent of those who show a rapid down-hill course in the first 48 hours after injury (see Cushing's observations [7]) A high CSF pressure is the rule in this latter group Most individuals with a subdural hematoma who develop a systolic blood pressure over 170 are likely to have an elevated lumbar puncture pressure (17 out of 18 cases where such combined studies were done) A high CSF (lumbar puncture) pressure, on the other hand in subacute or chronic cases is more often unassociated with a rise in blood pressure

Causes of Death. The mortality in cases of subdural hematoma when all groups are included ranges from 39 to 41 per cent in different series [14, 44]

The cause of death in those operated in the first week after injury and especially in those who come to surgery in the first 48 hours may be attributed largely to direct brain trauma A high per cent of the deaths among the subacute and chronic cases are in those

who have become comatose and who might have been saved by earlier operation It is very evident that if a patient is comatose or deeply stuporous before surgery his chances of survival are greatly lessened

In the subacute and chronic cases coma only too frequently is due to irreversible changes in the brain stem and elsewhere resulting primarily from compression by the hematoma [27, 86] which explains the lack of improvement following operation in many of the unconscious patients

At operation in some cases the compressed brain fails to "expand" after evacuation of the subdural clot and the mortality is high in this group

Surgical Treatment of Subdural Hematoma The timing of surgical intervention is extremely important The acute cases are emergencies and may be indistinguishable from epidural or intracerebral hematomas In the subacute and chronic group the drowsy patient must be followed with extreme care and operated on before the appearance of coma which often, in a drowsy patient, comes on suddenly

Local anesthesia supplemented with intravenous Pentothal Sodium, when necessary, is the most satisfactory method of anesthetizing these patients, especially when they are irrational, drowsy or stuporous

Perhaps the most satisfactory operative approach in patients suspected of having a subdural hematoma is *through the temporal region on each side with the* patient supine, the head supported by a posterior neck rest (or sand bag), and the head of the operating table elevated (Fig 5) Both sides of the head are explored as about 18 per cent of these cases have a bilateral lesion The head should be draped so that a large subtemporal decompression and occipital burr holes for

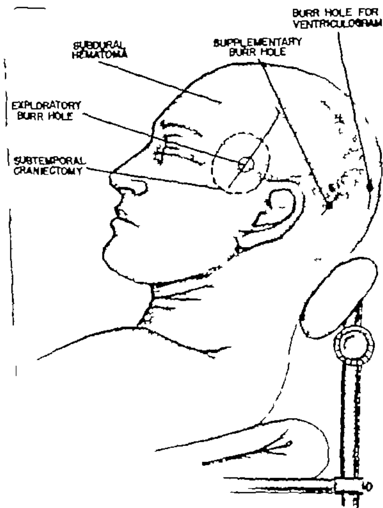


Fig. 5 Posit on of the patient and operative approach used in removing a subdural or epidural hemorrhage.

ventriculography can be carried out if necessary The great majority of subdural hematomas can be found and removed through the subtemporal approach al though additional burr holes may be made when indi

cated Obviously if a hematoma is exposed during the performing of occipital burr holes for ventriculography it may be evacuated through such an approach if it is fluid, or further burr holes may be required more anteriorly if there is any question of the existence of a solid clot

If a hematoma is not immediately exposed on opening the dura in the temporal region a curved instrument may be gently passed throughout the subdural space over the lateral surface of the hemisphere, keeping it close to the dura and avoiding the bridging veins near the longitudinal sinus and low in the temporal region When a semisolid clot or thick membranes are present the subtemporal decompression can be extended to any size desired and in a direction toward the center of the hematoma If the clot is large and semisolid (currant jelly type) it is often most easily removed by inserting the gloved finger or a curved instrument widely into the subdural space Some clots are relatively firm and loculated and this method is then more satisfactory than irrigating the subdural space with saline When membranes are encountered a large piece of both the outer and inner one may be removed for drainage through the decompression Sometimes fluid is present beneath the inner membrane and hence it should always be opened It has only occasionally been considered necessary to remove large portions of the subdural membranes It is well to drain all cases for 24 hours with a soft rubber drain inserted through a stab wound in the scalp The dressing will usually be found soaked with brown fluid on the first postoperative day

Among the advantages of the subtemporal opera

tion is that when a large opening must be made the decompression will be adequately protected by temporal muscle and fascia which is not true in operations in the posterior parietal region In addition with the patient supine and the head of the table elevated the contents of the hematoma will usually gravitate with ease out through the subtemporal opening Epidural and intracerebral hematomas subdural hydromas and areas of intracerebral softening in the temporal or inferior frontal region can also be evacuated through the subtemporal approach

In the series of 300 operated subdural hematomas referred to throughout this section it was necessary to turn a bone flap in only a small number of cases and this was usually to remove an organized liverlike clot or one with unusually thick membranes that appeared to prevent re-expansion of the compressed brain. Certainly in the sick patient with a subdural hematoma a *craniotomy should be avoided whenever possible* [42]

SUBDURAL HEMATOMA IN INFANCY AND CHILDHOOD

Subdural hematomas are common in infancy and childhood. A history of trauma is obtained in the minority of cases especially in infancy Malnutrition and a deficiency of vitamin C is present in many with signs and symptoms of scurvy in some patients. The bleeding and clotting time may be increased

The symptoms appear at any time after birth The infant becomes irritable and listless A convulsive seizure often jacksonian in type occurs in a high

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tolerate the performance of bilateral skull burr holes which should be made if fluid continues to form in the subdural space Such a procedure is also used for diagnosis in the older child where a subdural tap is not possible

The burr holes are placed in such a manner that the subdural space can be drained and a bone flap turned later if necessary If membranes are found bilaterally they are removed on only one side as thoroughly as possible through a bone flap About a week later a similar procedure is carried out on the opposite side and the membranes there excised

Comment The etiology and nature of many subdural hematomas in infancy and childhood appears to be different from that in adults Some systemic factor as mentioned, is often present, for instance malnutrition or even scurvy This factor seems to account for findings rarely seen in adults, such as retinal hemorrhages and fresh bleeding in the subdural space and into the cerebrospinal fluid even in chronic cases The hematomas as stated, very often consist of yellow fluid In adults it is true, the subdural fluid may be yellow but these cases are in the minority and not the majority as is true in infancy Furthermore a fluid subdural hematoma rarely reforms when removed in the adult even though the membranes surrounding it are allowed to remain In infants and children the subdural space usually rapidly refills after drainage unless the membranes are excised For this reason alone the membranes should be largely removed in the young patient In addition the rapidly enlarging brain of the infant may be hampered in its growth if a membrane which constricts it is left in place. In some infants fluid

percentage of cases The patient may vomit and over a period of days or weeks become progressively drowsy The temperature is frequently elevated in part as a result of dehydration

Examination may reveal a bulging or tense fontanelle with a large head and separation of the sutures in infants In older children roentgen examination may show separation of the sutures and other evidence of increased intracranial pressure Subconjunctival or retinal hemorrhages, are found in a considerable number of cases and internal strabismus when the intracranial pressure is high Papilledema occurs in some children where the head cannot enlarge with ease The deep reflexes are commonly increased or unequal and a positive Babinski is not infrequent Hemiparesis or even hemiplegia may appear

Lumbar puncture often reveals an elevated pressure and blood-tinged spinal fluid which is xanthrochromic on standing In other patients the spinal fluid is xanthochromic or clear The diagnosis in infants is confirmed by a subdural tap done through the most lateral aspect of the anterior fontanelle This tap usually obtains a yellow fluid or a bloody one that is yellow after standing The subdural or spinal fluid as a rule has an elevated protein The majority of these subdural hematomas are bilateral

Treatment of Subdural Hematoma in Infancy It is usually wise to remove no more than 15 cc of fluid from the subdural space on each side during the first tap Thereafter alternate sides of the head are tapped every other day removing 15 to 25 cc each time for one to two weeks During this interval the infant generally improves and begins to take its nourishment much better The patient is then well enough to

subdural one where motor weakness is generally less severe. A common history is that following trauma to the head a patient regains consciousness and subsequently becomes drowsy and develops a progressive hemiparesis. Exploration is carried out through the temporal regions for a suspected epidural or subdural hematoma but neither lesion is found On the side opposite the motor weakness evidence of brain injury may be present in the form of swelling subarachnoid hemorrhage contusion or laceration In any case it is wise to enlarge the subtemporal decompression and examine the anterior portion of the temporal lobe and the inferior posterior portion of the frontal lobe Not infrequently an area of softened devitalized cerebral cortex will be found Application of the sucker to this area may immediately lead one into a large intracerebral clot which can be removed with ease leaving a fairly smooth cavity At times it is necessary to enlarge the cortical opening and insert retractors to evacuate the clot adequately for some of them may be huge, especially when a branch of the middle cerebral artery has been lacerated by an overlying depressed fracture.

In some patients, as described by Botterell when a portion of destroyed brain is removed with the sucker in the manner mentioned above an area is entered which is not hematoma but macerated at times liquefied brain perhaps mixed with a small amount of clot. An area of softening of this type may act like an intracerebral blood clot and gradually enlarge by the acquisition of fluid and cause cerebral compression. When such an area is entered any straining by the patient may cause the devitalized brain tissue to be extruded like vomitus, again leaving a fairly smooth cav

does not reform following repeated tapping In these cases it is questionable whether any further surgical procedures are indicated

Of course where the etiology is traumatic and no complicating systemic element is present, subdural hematomas in infancy and childhood are much the same as in adults and then a true blood clot rather than yellow fluid may be found in the subdural space

An encapsulated collection of yellow fluid in the subdural space may also result from an inflammatory process such as a meningitis or an otitis media Treatment of these cases is the same as outlined for subdural hematoma

SUBDURAL HYDROMA OR HYGROMA

Occasionally a blow to the head will cause a tear in the arachnoid which allows escape of cerebrospinal fluid in ball valve fashion into the subdural space with little or no associated hemorrhage The fluid accumulates and may cause symptoms identical to those described for subdural hematoma[72]

INTRACEREBRAL HEMATOMA AND LOCALIZED INTRACEREBRAL MACERATION

Traumatic intracerebral hematomas have been estimated to occur in less than 1 per cent of severe head injuries[24] The signs and symptoms of such a lesion may closely resemble those reported for an epidural or subdural hemorrhage and differential diagnosis is not usually made preoperatively A hemiplegia is more common with an intracerebral clot than with a

4

SURGICAL LESIONS OF THE SCALP, SKULL AND BRAIN

LESIONS OF THE SCALP

Hematoma of the scalp in youth and in adults is usually not much of a problem The blood as a rule liquefies and is absorbed.

A scalp hematoma in infants likewise will generally be absorbed spontaneously Sometimes it enlarges temporarily and occasionally it persists for weeks giving anxiety to the parents.

If the hematoma is enlarging, unsightly or fails to absorb it sometimes should be aspirated. This is done carefully after proper preparation of the skin by inserting a needle well to one side of the hematoma and passing it through the pericranial tissues along the outer table of the skull until it enters the fluid collection Do not tap directly over the clot as the opening may persist and infection enter Pressure is applied to the needle track for many minutes after withdrawing the needle to be sure no bleeding occurs.

Cephalhematoma. Occasionally in the newborn a hematoma will form under the pericranium and strip it away from the skull. This pericranial tissue may ossify forming a new outer table of the skull over the

ity When no evidence of an expanding lesion is found a ventriculogram is usually indicated if the clinical picture has indicated the presence of such a lesion (For an excellent paper on intracerebral hematoma see Browder and Turney, 1942)

galea and well tied so that the wound edges are firmly approximated. This takes all tension off the skin in which the sutures should never be tied tightly. A dressing is applied and held in place with liquid adhesive or collodion about its edges.

A roentgenogram of the skull should be taken if there is any suspicion of a depressed fracture. Tetanus antitoxin or toxoid is used as in all wounds.

More extensive lacerations of the scalp are handled in the operating room in much the same fashion as described for the smaller wounds. The head is shaved widely and the scalp painted with antiseptic. Procaine, with epinephrine three drops to the ounce, is used to cause a block anesthesia and prevent hemorrhage. The wound is then copiously washed with soap and water, saline and some merthiolate followed by saline. During this stage bleeding is controlled with self retaining mastoid or thyroid retractors. All damaged tissue is debrided. Galea sutures of silk or catgut are very useful here to take all tension off the skin before the wound is closed (Fig. 6). Actual loss of scalp tissue can make closure complicated and may require ingenious swinging of scalp flaps (see under penetrating wounds).

FRACTURES OF THE SKULL

In this section the important types of skull fractures are discussed although some of them are not surgical lesions.

Simple linear fractures of the skull are of little significance in diagnosis, prognosis or treatment. They are evidence that a head injury has occurred, but give no indication as to whether or not there has been

surface of the clot and can be seen in a roentgenogram. In addition pressure erosion of the skull at times occurs beneath the hematoma.

Treatment consists of early aspiration and rarely of incision and removal of a solid clot. When actual bone formation takes place this must be exposed and removed.

Laceration. There is a popular belief that lacerations of the scalp rarely become infected and that in a hospital they should be treated by the most junior interne, usually in the accident room. Lacerations do become infected and this is a serious complication if there is an underlying depressed fracture, for then osteomyelitis, meningitis or brain abscess may follow. Should an operation for an intracranial hematoma become necessary, an infected scalp introduces a grave danger.

Small lacerations if handled properly may be treated in the accident room, but extensive ones are more safely cared for in the operating room.

In treating small lacerations, the scalp should be shaved for about two inches around the skin defect. The scalp is then cleansed with merthiolate or a similar solution and may be anesthetized with procaine. The wound itself is washed with soap and water and then swabbed with merthiolate followed by saline. The wound is draped with moist towels.

The depths of the wound are now carefully examined, explored with the gloved finger, and gently probed if necessary to rule out completely the possibility of a depressed fracture. The skin edges are debrided with scissors, the wound is again washed with saline and closure carried out. If there is active bleeding one or more sutures are used through the

Simple depressed fractures are treated surgically in adults unless the depression of the inner table is only a few millimeters in depth Clinically they may be palpated through the scalp but this method can be deceptive. A hematoma of the scalp rather commonly will have a soft center and firm edges which to the palpating finger gives the definite impression of an area of depressed skull Roentgenograms are necessary for diagnosis and for deciding the type and location of the skin flap that is to be turned down in exposing the fracture. When the depressed area is in the frontal region the skin incision should be along the line of the coronal suture within the hair line The operative procedure otherwise is as described below for compound depressed fractures of the skull The chief difference is that when the fracture is not compounded the bone fragments after being removed are usually thoroughly washed and replaced.

Depressed Fractures in the Newborn. Simple depressed fractures are common at birth and are often associated with an extensive indentation of the lateral aspect of the skull. At times an indentation is present without any actual fracture and in some cases can be corrected by manipulation of the skull

The depressed skull must be corrected promptly and this requires surgery especially if a fracture is present. In these cases the area of depression unlike those in adults, must be truly elevated. The procedure is simple A burr hole is made in the skull and the bone elevated everywhere to its normal contour with a flat director passed between the dura and the skull

Compound Fractures. The ordinary compound

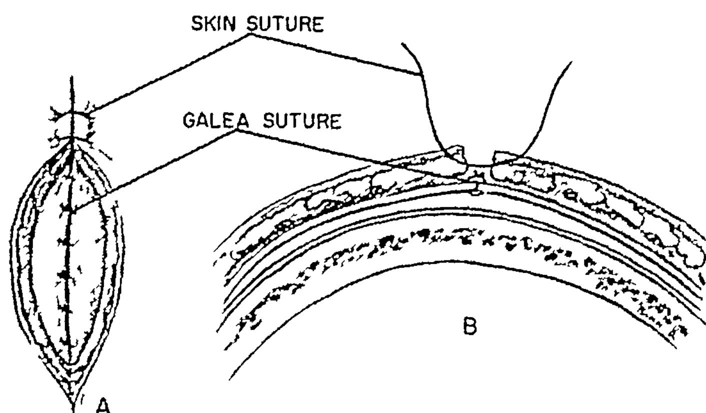


FIG 6 Galea and skin sutures used in closure of the scalp

intracranial injury They require no treatment in themselves and will heal in about six months in children and at a later date in most adults Presumptive but not absolute evidence of fracture is the presence of a periorbital hematoma following a blow to the back of the head, ecchymosis behind the ear, blood in the middle ear causing discoloration of the drum, drainage of blood from the nose or ears, or edema extending in linear fashion over the lateral aspect of the skull Escape of cerebrospinal fluid from the nose or ears, of course, means that there is a fracture Bleeding from the ear may occur in the absence of fracture as a result of laceration of the ear canal following a blow to the jaw

What has been said of simple linear fractures applies equally to simple comminuted fractures If the latter are extensive one may surmise that the blow has been quite severe but little more

The head is shaved and the wound prepared anesthetized and draped as described for extensive lacerations and for penetrating wounds. The lacerated area is excised and the wound extended with a straight or curved incision to expose adequately the area of depression (Fig 7) Self retaining retractors, Michel clips or hemostats on the galea are employed to control hemorrhage. With the common depressed fracture seen in civilian life that occurs from a blunt instrument like a hammer or larger object a small trough of bone is removed entirely around the periphery of the depressed area To start this removal usually requires the making of a burr hole to one side of the depressed region (Fig 8A and B) but at times this is unnecessary as a purchase with the rongeur may be possible on one of the bone fragments. After removing the trough of bone as described (Fig 8C) all the depressed fragments can then be extirpated almost simultaneously Any underlying vessel on the dura or brain which bleeds when the pressure of the bone is removed can now be promptly controlled with silver clips (Fig 8D and E) coagulating current Gel foam or fibrin foam Do not elevate or withdraw any bone fragments until it is possible to remove almost all the depressed area rapidly at the same time or bleeding may be started beneath this area and cause severe damage to the brain before the remaining fragments can be rongeured away and the offending vessel exposed.

It should be noted that except in children compound depressed fractures are very rarely elevated. The bone fragments are removed and usually discarded although they may be washed and in some cases replaced Little concern need be given to the

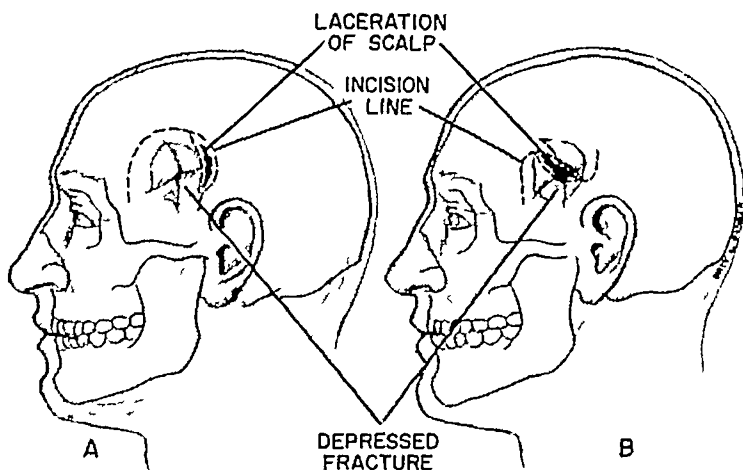


Fig 7 A and B Methods of débridement of a scalp laceration and exposure of a compound depressed fracture of the skull

linear fracture of the skull passing beneath an area of lacerated scalp requires no treatment except cleansing and debridement of the wound as described

A simple linear or even slightly depressed fracture into the paranasal sinuses is best treated with sulfa drugs and antibiotics, unless complications arise

Compound depressed fractures occurring over the vertex or convexities of the skull should be treated surgically The hazards of meningitis, brain abscess, brain damage, and post-traumatic epilepsy are too great to handle them otherwise

The diagnosis is made by inspection of the wound and by roentgenogram The laceration of the scalp frequently lies at some distance from the underlying fracture and this must be considered carefully in planning the draping of the head and the scalp incision to uncover the depressed area (Fig 7A and B)

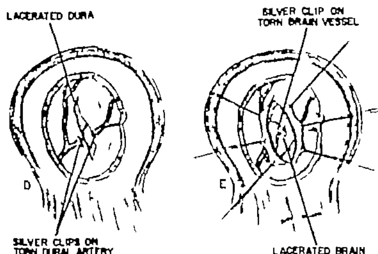


FIG 8 (Cont)

When the underlying dura and brain have been exposed the first concern is control of hemorrhage A large piece of moist gauze or brain cotton can be quickly placed over the exposed area and hemorrhage controlled with gentle pressure The gauze is removed in stages and each bleeder handled with clip or coagulation as it is exposed As mentioned when bleeding is present between dura and skull the dura can be sutured to the periosteum thus tamponing it against the inner table of the cranium If the dura is intact a tiny area usually should be opened especially if it appears blue to rule out a subdural hematoma If the dura is already lacerated the exposed brain is inspected carefully and any grossly macerated portion gently removed with the sucker Macerated brain may be not only a source of infection but of later scar formation Obviously subdural or intracerebral hematoma also should be evacuated To

fact that a skull defect will be left as this can be covered by a plate at a later date

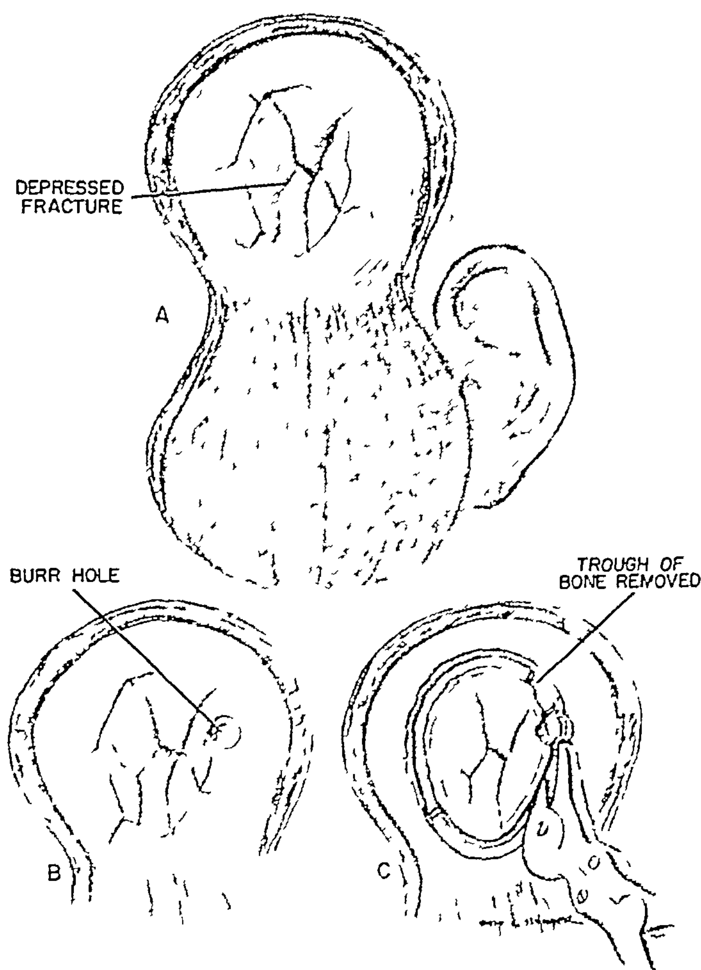


FIG 8 Stages A B and C in removing an area of depressed skull fracture Bone fragments should not be disturbed until Stage C has been completed Control of bleeding D from middle meningeal vessel and E from brain vessel after removal of depressed skull fragments

there are signs of neurologic deficit. In treating more severe and possibly contaminated depressed fractures involving a sinus expert surgical handling is required. At least 1000 cc. of blood should be available.

As described, in any depressed fracture fragments of bone should not be removed until the entire depressed area can be elevated at the same time. If a tear in the sinus is encountered it should be covered immediately with brain cotton and compressed with the finger to prevent air entering. Preparation is then made to close the opening. If the size of the tear is small this may be sutured, or a piece of muscle can be sutured over it. When a shelf of intact bone lies above the torn area muscle or Gelfoam can be placed over it and the dura sutured to the pericranial tissues, thus tamponing the hole in the sinus as is done so often to control dural bleeding in any craniotomy. When a dural sinus is torn completely across it should be securely ligated or closed with silver clips.

PENETRATING WOUNDS OF THE BRAIN

Our knowledge of the treatment of penetrating wounds of the brain has been derived chiefly from war experiences,[2, 22, 42, 3, 43, 44] although such injuries are not uncommon in peacetime. The basic principles involved in the handling of these cases as laid down by Cushing in World War I remain applicable today but the use of the sulfa drugs, penicillin and emergency transfusions as well as earlier treatment of the wound by the neurosurgeon in World War II has led to certain changes in management and has lowered the mortality rate.

Classification of Penetrating Wounds. Although

the dura is then closed tightly using, if necessary, a periosteal graft, temporal fascia or fascia lata

Penicillin or sulfa powder should not be used on the brain for fear of causing convulsive seizures It is best to leave a thin rubber drain through a stab wound in the scalp for 24 hours

Compound Fractures into the Frontal Sinus A compound depressed fracture of the frontal region involving the frontal or ethmoid sinuses, if extensive, or if it includes the posterior wall of the frontal sinus where the dura may be lacerated, should be treated surgically The area of involved bone can be approached under local or general anesthesia through the skin laceration or, if this is small, through a long coronal incision turning the skin flap forward over the face The frontal sinus usually should be completely obliterated down to the frontonasal duct, removing all mucous membrane and the entire posterior wall of the sinus The duct can be covered with a small piece of Gelfoam The dural and brain wound are handled as already described and the dura closed tightly A loose packing and sterile catheter or soft rubber drain may be left in place and brought out through a stab wound to one side of the eyebrow Daily irrigation with penicillin or bacitracin through the catheter can be used The packing usually is entirely removed in several days Antibiotics and sulfa drugs are best continued for a week or more

Compound Depressed Fractures Involving the Longitudinal or Major Venous Sinuses[41] Small slightly depressed compound fractures over a venous sinus due to a blow from a blunt instrument are at times best left alone except for superficial debridement, unless

there are signs of neurologic deficit In treating more severe and possibly contaminated depressed fractures involving a sinus expert surgical handling is required. At least 1000 cc. of blood should be available.

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Classification of Penetrating Wounds. Although

there is an infinite variation in penetrating brain wounds they may be satisfactorily divided into four groups

1 Wounds in which an instrument like a knife or icepick is thrust into the brain

2 Wounds in which bone fragments are driven through the dura into the brain as in the gutter type of bullet wound, or in the common depressed fracture resulting from a hammer blow

3 Wounds where in addition to pieces of bone, a bullet or shell fragment has lodged in the brain

4 Wounds of through and through type, with a point of entry and of exit, where the missile passes directly through some portion of the intracranial cav ity

Signs and Symptoms and Indications for Treatment A practical classification of the early signs and symp toms is one based on the severity of the injury[106] which, of course, will depend upon many factors in cluding the type of penetrating missile and the func tion of the area of brain damaged A patient may be critically injured, seriously injured or less seriously injured An assessment of the severity of the condi tion will determine whether the patient can tolerate surgery and, if so, how urgently it is indicated

The critically injured are those who appear more or less moribund from associated wounds to other parts of the body damage to the vital centers of the brain, or brain compression from intracranial hemor rhage and brain maceration In this group nice judg ment is required in selecting patients for surgery Those in shock, especially from hemorrhage or asso ciated injury, should be given immediate care At tention to fractures, chest or other wounds, may im

prove the patient's condition to the point where the intracranial injury can be assessed and treated surgically when indicated. It seems established that there are few operative neurosurgical emergencies among cases of penetrating wounds of the brain and that actually the critically or seriously injured may usually be benefited by 12 to 24 hours of conservative treatment. On the other hand when signs of severe brain compression from probable intracranial hemorrhage are present emergency surgery is indicated provided the patient's condition does not appear hopeless.

The seriously injured patient with a penetrating wound of the brain is one in whom the wound is serious, but the general condition and vital signs do not suggest immediate danger to life. In these cases surgery should be performed as soon as proper facilities are available They should be treated as emergencies, especially if the condition begins to deteriorate apparently as the result of intracranial hemorrhage

The less seriously injured are those with penetrating brain wounds that have not resulted in any serious neurologic or general physical abnormality These patients are conscious and may be ambulatory Some of them will have insignificant scalp wounds through which a piece of sharpnel has penetrated the brain without in some cases, causing a loss of consciousness. Such cases illustrate how essential it is never to overlook or underestimate the seriousness of the smallest scalp wound Surgery may be postponed temporarily in many of this group

Surgical Treatment of Penetrating Wounds The present treatment of penetrating wounds of the brain is well established Treatment at or near the

place of injury should consist of emergency care of shock and of hemorrhage, if necessary The head wound must not be tampered with except perhaps to shave and cleanse the surrounding scalp, control sur face bleeding and apply a dressing Tetanus anti toxin and antibiotics are given if there is to be some delay in definitive surgery

It has been shown that patients with penetrating wounds of the brain stand transportation very well unless they are in shock They should be taken to a hospital where modern neurosurgery with all the nec essary equipment is available In wartime this was usually the evacuation hospital five to 20 miles be hind the front line These wounds must be treated as Cushing stated *in an "all or none fashion"* In complete operations must never be done

The general management and surgical approach is much the same as described for the ordinary com pound depressed fracture Adequate stereoscopic, pre operative roentgenograms of the skull are taken and ample blood for transfusion must be available The head is completely shaved and operation carried out under local anesthesia if the patient is co operative Otherwise endotracheal ether is used

Before draping the head, the skin incision to be employed must be carefully considered The old tripod incision used in World War I has been largely abandoned The area of lacerated scalp is excised and the incision either extended in linear fashion or curved around the periphery of the area of involved skull so that a flap can be turned (Fig 7A and B) *This flap can be rotated at the time of closure to cover an area of skin defect if necessary When a scalp de fect is left it should be over a normal area of skull*

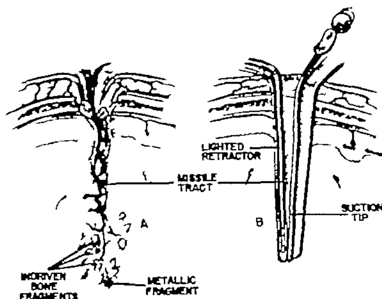


FIG 9 A and B Débridement in a case of penetrating wound of the brain.

and this should be skin grafted immediately if possible usually with a split thickness graft

Thorough débridement of the wound must be carried out. All devitalized soft tissues and all bone fragments are removed as described for compound depressed fractures. The cranial defect is enlarged until normal dura is everywhere exposed. The dura is débrided excising as little as possible. The brain itself is now carefully debrided removing all macerated brain and bone fragments with the aid of gentle suction (Fig 9A and B) The tract into the brain is followed under direct vision to its depths. If infection is to be prevented *it is essential that all bone fragments be obtained* with if necessary the help of additional roentgenograms, and this is true also of all

5

OTHER SURGICAL COMPLICATIONS FOLLOWING HEAD INJURY

WOUND INFECTION

Wound infection has always been one of the most dreaded complications of head injury. Fortunately this complication is now not so common and usually not so serious since the introduction of modern neurosurgery and the use of the sulfa drugs and antibiotics.

In closed head injuries meningitis can occur as a result of a fracture through a paranasal sinus or into the middle ear but brain abscess is rare. Very occasionally after a contusion of the scalp an infection sets in leading to osteomyelitis of the skull which may be followed by an abscess in the epidural or subdural space or within the brain.

Infection in open wounds of the scalp, cranium or brain is more common.

Infection in a simple laceration of the scalp is not usually a serious complication unless intracranial surgery through this area is contemplated. When it occurs and is manifested by reddening and tenderness of the skin edges, the head is shaved rather widely

Diagnosis of Deep Infection Deep infection and even chronic brain abscess formation may take place with few symptoms and signs especially when antibiotics have been used. Early symptoms include headache and stiff neck. The latter may be absent. Headaches are nearly always present but may be somewhat delayed and come on gradually The decompression may cease to pulsate and become tense Papilledema commonly is present accompanied by neurologic signs of an expanding brain lesion Signs of meningitis can appear at any time, especially during the first three weeks after injury through spreading of the infection into the ventricles or subarachnoid space.

Treatment of Deep Infection It has been demonstrated[92] in the past war that when deep infection occurs in a penetrating wound debridement or redebridement should be carried out without delay True *abscess formation* may not yet be present but it is deemed unwise to wait in the hope that a capsule will form In most cases roentgenograms will reveal retained foreign bodies and these will be found to lie in a pocket of pus with varying amounts of necrotic brain along the missile tract. All pus foreign bodies and devitalized brain are removed with the sucker and the dura and scalp closed without drainage. When unhealthy granulation tissue is present beneath the scalp it is usual to drain the wound for a few days

Chronic Brain Abscess In some patients a posttraumatic brain abscess is treated in the same manner as used in nontraumatic cases.[84–86] This is especially true if no foreign bodies remain within the brain If the abscess is chronic and a fairly thick capsule has formed the abscess may be excised.[88–91]

the skin sutures removed and moist dressings applied. If there is purulent drainage, the wound should be packed loosely open and local bacitracin, sulfathiazole or other ointment used. The appropriate antibiotic can be given after testing the organism's sensitivity, if desired.

Infection Following Penetrating Wounds

Superficial infection can be a very serious affair when it takes place as a complication following craniotomy or craniectomy performed in the treatment of intracranial hematoma or of a penetrating wound. Such an infection most often is due to improper scalp closure. The infection may remain in the scalp and linger on for weeks causing one galea suture after another to slough out. On the other hand, and especially if the dura is left open, the infection may extend inward causing an osteomyelitis, a meningitis or an intracranial abscess. Treatment will vary with the circumstances. Hot, wet dressings, antibiotics and removal of sutures may be sufficient if the infection is confined to the skin.

Low-grade infection in the extradural tissues may persist. In such a situation if a bone flap, skull plate or extradural bone fragment is present it usually has to be removed before healing will occur.

Deep infection in the epidural or subdural space or within the cerebral hemisphere may follow any inadequately treated compound depressed fracture with lacerated dura and brain. In penetrating war wounds deep infection has been found to be ten times more frequent when a foreign body, especially a bone fragment, has been left within the cerebral hemisphere.[63]

injuries has been described in Chapter 2 The same general treatment is used when meningitis develops in a patient with a penetrating wound of the brain In addition the local source of the infection which is often a retained foreign body is generally treated surgically

Cerebral fungus[44] or in other words a herniation of brain tissue through an opening in the skull may follow operation for an intracranial hematoma or penetrating wound. It can be of two types a *benign* fungus which is the result of local cerebritis wound infection and an open dura, or a *malignant* fungus due to increased intracranial pressure caused by deep infection brain abscess, hematoma or rarely necrotic brain tissue. In the benign type hot wet dressings and antibiotics are used and the fungus usually will heal With the malignant form the hernia is progressive until the cause is attacked directly and eradicated.

Osteomyelitis of low-grade localized type may follow any compound depressed fracture or intracranial operation. It can usually be handled with ease by removal with rongeurs Only occasionally does the disease process spread to involve a large area of skull

CEREBROSPINAL FLUID RHINORRHEA

Drainage of cerebrospinal fluid from the nose or rhinorrhea (see Cerman 1911) has been mentioned in Chapter 9 Nonsurgical Cases It may occur as a result of a fracture involving the frontal ethmoid, or occasionally the sphenoid sinus. For such drainage to take place there must be a communication between the subarachnoid space and the paranasal sinus

If the lesion is less chronic the abscess can be drained by tapping and injected with 10 to 20,000 units of penicillin[105] on one or more occasions and then excised Antibiotics and usually sulfadiazine are used parenterally in all these cases, but as mentioned, there is a danger of convulsive seizures if penicillin is used on the brain after excising the abscess I have seen a psychosis develop during irrigation of the brain with penicillin following excision of a temporal lobe abscess

Meningitis Meningitis may occur as an occasional complication of a closed head injury when there is a fracture involving a paranasal sinus or the middle ear Meningitis is common in cases of penetrating wounds of the brain

Diffuse infection of the subarachnoid space has a habit of coming on suddenly This is particularly true in closed head injuries where the patient may become rapidly comatose over a period of 24 hours If the diagnosis is not made early it may be too late to save the patient However, prompt treatment usually produces a dramatic recovery The cardinal symptoms and signs are headache, drowsiness, high fever, stiff neck and other signs of meningeal irritation A case of meningitis can be overlooked in patients with acute trauma to the head since they so often have a stiff neck and high fever in the absence of meningitis However, when a patient who has been doing fairly well develops a sudden rise in fever, meningitis should be immediately ruled out and especially if drowsiness or signs of meningeal irritation appear

The treatment of meningitis in patients with closed

SKULL DEFECTS AND THEIR REPAIR

Repair of skull defects or cranioplasty is frequently necessary in cases of head injury following surgery or after treatment of compound fractures or penetrating wounds The skull defect may be covered with bone tantalum * or a number of other substances that cause little adverse tissue reaction

Small defects say the size of a 25-cent piece, usually do not require repair except for cosmetic reasons and will become firmly covered by fibrous tissue. Larger defects may bulge when the patient stoops over and if associated with headaches dizziness or other incapacitating symptoms probably should be repaired. Some patients develop apprehension and other psychological disturbances because of a skull defect which they believe renders their brain susceptible to injury If they cannot be reassured on this point repair is often helpful and of course is indicated when the patient's occupation is such that the area of skull defect might be injured On the other hand many patients will remain well and asymptomatic with large cranial defects.

There are many methods of making a skull plate and also of inserting it.[34] The plate may be made preoperatively say by the dental department as in the last war This method is useful in complicated plates where for instance a frontal bone with supra orbital ridges must be reproduced The average plate can be made with ease by the surgeon. Preoperatively a piece of paper is laid on the shaved scalp and with a pencil the outline of the defect is drawn on the paper making the drawing everywhere about one-quar

which implies a laceration of the arachnoid and the dura near or at the site of the fracture. Because of this intracranial communication there is always danger of a meningitis. In addition air may be driven in ball-valve fashion up through the nose and fistula and collect in the subdural space, within the brain, or even in the ventricles. Such a collection of air is referred to as an aerocele, is visualized by roentgenograms of the skull and at times produces signs and symptoms of an intracranial expanding lesion.

The initial treatment of rhinorrhea is usually conservative as already described and most cases will clear up within about one month. If the rhinorrhea persists, or if meningitis or aerocele appear it is then advisable to obliterate the fistula by intracranial closure of the dura. An endeavor is made to localize the site of the fracture roentgenographically and a rhinologist attempts by direct vision to see from which sinus the drainage is occurring. The operative approach may be unilateral through a transfrontal craniotomy. If the cribiform plate is involved a bilateral frontal flap is used. The fistula can be approached either extra or intradurally. It is excised and the dura closed with, if necessary, a fascial graft. Gelfoam may be placed over the fracture site extradurally. Any aerocele present will be spontaneously absorbed, but if it is under tension it can be tapped with a ventricular needle.

When a compound depressed fracture of the frontal region with or without laceration of the overlying scalp is present and accompanied by a rhinorrhea, immediate surgical repair is indicated. The compound fracture is treated as previously outlined and the fistula closed.

merely laid over the defect and fixed in place with tantalum wedges In certain regions to prevent slipping, or where the skull is thin a portion of the plate can be sutured to the soft tissues or occasionally to bone after making drill holes This simpler method of inserting the plate usually avoids exposing the dura and stripping it from the skull which is necessary when drill holes are made for wiring

Plates have been inserted at the primary operation for depressed fractures and other contaminated wounds but it is probably best to wait six months before performing cranioplasty in such cases. In clean operations a skull defect may be covered immediately

Postoperatively infection is uncommon after cranioplasty but sterile fluid may collect around the plate, sometimes for weeks requiring aspiration and the application at times of an elastic bandage to the head.

Arteriovenous Aneurysm. Occasionally a fracture of the skull will cause a tear in the carotid artery as it passes through the cavernous sinus As a result the arterial blood is forcefully pumped into the venous sinus. There follows venous congestion and back pressure with edema of the periorbital tissues on the involved side. The eye is gradually and usually progressively pushed outward at times leading to extreme exophthalmos, swelling of the conjunctiva and even corneal ulceration The extra-ocular muscles become edematous and all or some of them cease to function. The patient may or may not hear a bruit with each heart beat and careful inspection commonly reveals a simultaneous pulsation of the eye. On auscultation over the eye or temporal region a definite bruit is heard and this usually will disappear on compression of the carotid artery on that side When the stetho-

ter to one-half an inch larger than the actual defect A piece of tantalum is then cut with scissors to the size of the drawing Several newspapers or towels are laid on a hard surface and on this the piece of tantalum is pounded with a round-headed steel or wooden hammer The metal is malleable and with a little practice can be beaten to any shape desired in a few minutes At intervals it is laid on the patient's scalp for comparison with the contour of the surrounding skull and treated until it has the correct form

At operation it is generally preferable to explore the defect by excising the old scar in the scalp if this lies over the defect The incision may then be extended to any length desired or curved about the margins of the defect Adequate exposure is essential At times, especially in the frontal region, it may be preferable to turn a skin flap

There are several methods of inserting the plate The bone edges may be exposed and a shelf cut in the outer table so that the plate may be inlaid [30] The plate is then held in position until tantalum wedges, used like glazier's points, are driven into the bone around the margin of the plate

The plate may also be wired to the skull through drill holes in the bone about the periphery of the cranial defect

A much simpler, more rapid and highly satisfactory way of inserting the plate is as follows With a scalpel the periosteum is incised around the margin of the defect and about one quarter inch away from it It is then brushed backward with a periosteal elevator for a short distance The hand made plate, fashioned preoperatively or at operation as described, is then

Following ligation of the internal carotid artery the bruit may disappear only to return the next day After about 48 hours it may again disappear and this time permanently apparently as a result of thrombosis within the internal carotid which extends upward far enough to obliterate the fistula into the cavernous sinus. With the disappearance of the bruit the signs gradually recede and the patient is cured. Unfortunately only too often the bruit persists and it is then necessary to ligate the carotid artery intra cranially as well as in the neck.

ARACHNOIDAL CYSTS

Patients who have sustained a fracture of the skull with an underlying laceration of the dura and brain occasionally develop an arachnoidal cyst. Spinal fluid escapes through the laceration and at times even extra cranially In time collections of fluid become surrounded or encapsulated by outgrowing arachnoid forming a cyst Overlying the cyst the bone along the fracture line sometimes atrophies leaving a skull defect with irregular scalloped edges In the roentgen ray films the fracture will be seen running into this characteristic defect.

If symptoms are present or the condition is progressive operation may be indicated at which time the cysts are excised and the dura closed

POST-TRAUMATIC EPILEPSY

After a head injury a convulsive seizure may occur during the first few days or weeks or be delayed until sometime in the future when the brain has recovered from the acute effects of trauma When late seizures

scope is placed over the eye the eyelid on the opposite side should be closed gently with the examiner's finger. Otherwise flutter of the eyelid may simulate a bruit.

Roentgenography of the skull may show a fracture. Carotid angiography is useful in confirming the diagnosis. Usually the injected Diodrast is seen in the carotid artery up to the region of the fistula where it enters the venous system.

Treatment of Arteriovenous Aneurysm. Before performing any surgery it is well to test and at the same time improve the adequacy of the collateral circulation by compressing the involved carotid artery in the neck for gradually increasing intervals during about one week or longer. This may be done digitally at first or with a mechanical apparatus that can be left in place. Occasionally the fistula will heal spontaneously after a course of intermittent carotid compression.

Usually surgery is carried out at the end of a week, or sooner if the condition is progressing in an alarming fashion. There are several ways of approaching the problem. A satisfactory procedure is to obliterate the implicated common carotid artery in the neck with a home-made tantalum clip. About one week to ten days later the internal carotid artery on that side is also closed with a tantalum clip or Selverstone clamp. The procedures are done under local anesthesia and the patient's motor power and state of consciousness tested for a half hour or more before closing the wound. During the next week similar testing is carried out at very frequent intervals, so that the clamp may be removed immediately from the artery if any sign of weakness appears. The bruit should also, of course, be listened for.

considered to have an associated cerebral contusion as the probable cause of the seizures.

Brain injury during birth is a most important cause of epilepsy occurring later in life. In the process of delivery the brain can be injured by local trauma, compression or ischemia. A variety of brain lesions may result from birth injury including meningocerebral cicatrix, areas of cortex where the gyri are small and shrunken (microgyri), and more extensive lesions of a cerebral hemisphere. One of the most important epileptogenic lesions is scarring of the medial and inferior portion of a temporal lobe[64] apparently resulting from herniation of this area over the edge of the tentorium on excessive molding of the head during birth.[21]

Surgical Treatment. The problem of surgical treatment in cases of epilepsy is too extensive a one to be considered here *except in a superficial manner*. The outstanding works of Penfield[59, 64] and his associates should be consulted for an understanding of this most important subject.

Surgery is used in the treatment of certain carefully selected cases[60, 64] that have failed to respond satisfactorily to medical treatment and the appropriate use of anticonvulsive drugs.

The selection of patients for surgery is a complicated one and includes many individual factors in each case. The usual basic criteria required before exploring a patient for an epileptogenic lesion are evidence in the history and on observation that the attacks have a focal origin in the surgically approachable cerebral cortex or in the anterior or medial portion of the temporal lobe[5, 60] and confirmatory evidence in the electroencephalogram and in air studies

appear they tend to be recurrent Until further figures are available it is difficult to estimate with what frequency patients who have had seizures within several weeks following a head injury will later develop epilepsy

The incidence of late epilepsy following penetrating war wounds of the brain is high, having been estimated by Ascroft [3] as occurring in 23 per cent of patients in whom the dura remained intact and in 45 per cent of those with dural penetration In gunshot wounds as a whole, the incidence of epilepsy reported in one series of 500 cases [31] from World War I was 9 8 per cent, but this rose to 18 9 per cent where the dura was penetrated, to 26 1 per cent with retained metallic fragments and 66 7 when bone was retained

The occurrence of seizures either early or late is very low in cases of closed head injury in general, being reported as much less than 1 per cent by Elvidge [20] On the other hand in a series of 300 subdural hematomas with closed head injuries, 60 had a convulsive seizure before operation, an incidence of 20 per cent, [24] 56 of the patients with seizures had acute or subacute subdural hematomas associated in most cases with recent severe brain injury and cerebral contusion or laceration It seems more than likely that the brain contusion or laceration rather than the subdural clot was the cause of the seizures in these cases If this is so then early convulsions may be as frequent in closed head injuries as in penetrating wounds providing there is brain contusion or laceration These findings are in keeping with those reported in another series [60] of 71 patients with subdural hematomas, 25 per cent of whom had one or more convulsive seizures Most of this group were

6

SPECIAL AIDS IN DIAGNOSIS

TECHNICS

Lumbar puncture is a simple procedure and can provide much valuable information if properly performed. When incorrectly used or carried out it can be harmful and supply misleading data

The patient should be on his side near the edge of the bed and his spine flexed by drawing the knees into the abdomen and gently flexing the neck A pillow is used under the head to avoid lateral flexion of the neck and compression of the jugular vein. An unsterile towel is placed beneath the patient to protect the bed from the colored antiseptic solution used on the skin of the back. With the thumb nail the interspace between the spinous processes of the third and fourth lumbar vertebrae is marked with a cross directly in the midline. This interspace lies just above the pelvic brim The interspace immediately above or the two below may also be used if desired.

Sterile gloves are now used and the back widely painted at the level chosen with merthiolate or other antiseptic. Do not use drapes. A few drops of 1 to 2 per cent procaine can be used in the skin A 19-

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gauge lumbar puncture needle is inserted at the spot marked, after checking the position of the spinous processes The point is inserted so that it just misses the tip of the spinous process below it Holding the needle so that the shaft can be well visualized it is slanted slightly upward, about 30 degrees, and advanced slowly and steadily forward, keeping it in line with the longitudinal axis of the spine and on the same plane as the spinous processes Advancing the needle in jerks is painful Usually the point can be felt to pop through the dura It is advanced another one eighth to one fourth of an inch The stilette is withdrawn and immediately replaced if fluid escapes If no fluid is obtained the needle is advanced a little further in stages, each time withdrawing the stilette, until fluid escapes If the needle strikes the anterior wall of the canal it is withdrawn in stages If still no spinal fluid is obtained the procedure must be repeated

When fluid escapes from the needle be careful not to lose more than a few drops Apply the water manometer to test the lumbar puncture pressure Now relax the patient's legs and extend them a little so that he is not straining in any way Have the arms lie loosely on the bed Ask the patient to relax completely, close his eyes, and breathe in and out with his mouth open Usually the cerebrospinal fluid pressure will be noted to fall gradually until it reaches the correct reading for that patient This may take many minutes The normal pressure is anywhere from about 70 to 160 Any straining whatsoever will give a false reading Now remove the desired amount of fluid slowly and take a final reading

If a lumbar puncture is done always take a pres

sure reading Don t try to estimate the pressure by the rate of escape of the C.S.F This is grossly in accurate Remove a few cubic centimeters of fluid into each of three test tubes In this manner an eval uation can be made as to the origin of any blood which may be present. If the lumbar puncture itself has caused the bleeding the first tube will contain the most blood. If the blood came from intracranially all the tubes will be the same color or the third one may be a deeper red

Never do a lumbar puncture on a patient with an acute head injury if he struggles and resists and never compress his jugular veins to perform a Quechenstedt test. Either procedure may produce a harmful rise in intracranial pressure and straining may result in a rise in blood pressure and consequent intracranial hemorrhage

Pneumo-encephalography was introduced by Dandy in 1919 In this diagnostic test the cerebrospinal fluid is withdrawn and replaced by air or other gas The air fills the ventricles and subarachnoid spaces of the brain and will thus outline them in roentgenograms taken of the skull

A pneumo-encephalogram is performed with the patient in the sitting position The head is flexed slightly forward and the arms supported in front of the patient on a rest A sheet is tied around the thorax and beneath the axillas and then about the chair for support when an anesthetic is used. Some use local anesthesia and sedation The patients have much less pain react better and have less shock if intravenous thiopental sodium is used

A lumbar puncture needle is inserted at the third or fourth lumbar interspace and a pressure reading

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taken When normal the fluid rises in the manometer to the level of the patient's neck, usually around 450 mm A three-way stopcock is left on the needle Before removing any fluid 10 cc of air are injected after drawing it into a 10 cc syringe through a piece of sterile gauze Always before injecting any air be sure there is a very free flow of C S F under a fairly high pressure (i e, around 450) Now alternately withdraw 5 cc of C S F and inject 5 cc of air until about 90 to 110 cc of air have been used If there is some elevation of the spinal fluid pressure, or if a brain tumor is suspected, smaller amounts of air generally are used It is a useful practice to take one anteroposterior roentgenogram of the skull after 30 cc of air have been injected to obtain an estimate of the size of the ventricles and to determine whether air is entering them In cases of hydrocephalus or cerebral atrophy, more air may be used than usual, providing fluid is obtained Test the pressure at intervals If it falls below its initial level small amounts of extra air may be injected to maintain the original pressure

Following the injection of the air the patient's head must be kept in such a position that the gas cannot escape from the ventricles He is now taken to the Department of Roentgenology Proper manipulation of the head during the taking of the roentgen ray films is necessary to obtain maximum filling of the ventricles with air

Pneumo-encephalograms are not usually done on patients if the intracranial pressure is much above normal, on patients with more than one diopter of papilledema, nor on those who may have a posterior fossa tumor Ventriculograms are used instead in these

cases. In a patient suspected of having an intra cranial surgical lesion the air study is performed in the morning and the surgeon must always be pre pared to operate the same day if an expanding lesion is demonstrated Otherwise a chain of events may be precipitated leading to the patient s death

Ventriculography first performed by Dandy in 1918 is done by injecting air directly into the lateral ven tricles through occipital burr holes.

INTERPRETATION OF PNEUMO ENCEPHALO GRAMS AND VENTRICULOGRAMS

Air studies are of value in nonsurgical cases in in dicating the presence of brain atrophy or injury In surgical cases these methods are very useful in diag nosing certain cases of intracranial hematoma or ab scess formation (Fig 10A–D)

Pneumo-encephalography in Nonsurgical Cases After a severe trauma to the head an air study not in frequently reveals some dilatation often asymmetrical of the ventricles (Fig 10D) In addition there may be increased amounts of air in the subarachnoid spaces over the cerebral hemispheres These findings are usually interpreted as evidence of brain atrophy which may be more marked in the cortex or in sub cortical areas. If injury to one cerebral hemisphere is severe the lateral ventricle on that side may be larger than the opposite one and the entire ventricu lar system shifted toward the atrophic hemisphere

At times a collection of air will be seen filling a "cyst within the cerebral hemisphere, which com municates with the ventricle or subarachnoid space. Such an abnormal collection of air is due to the de

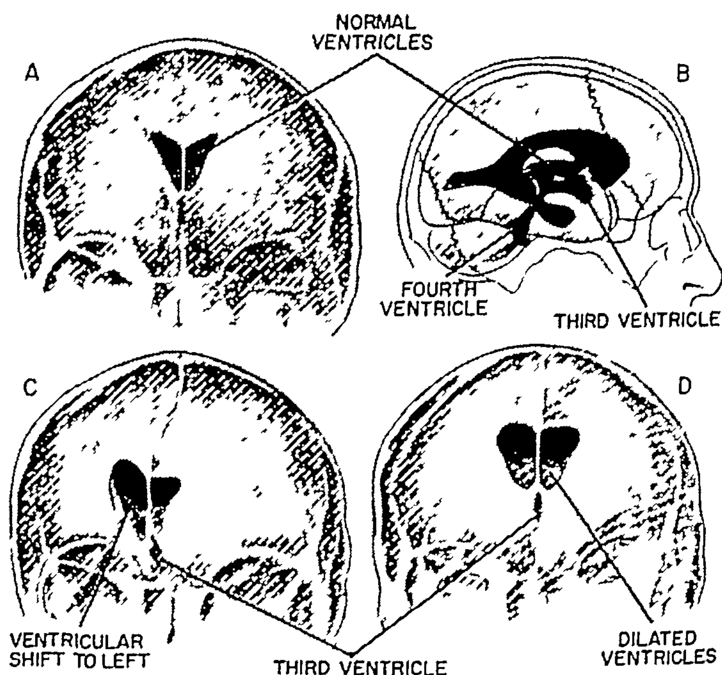


FIG 10 Appearance of ventricles after ventriculography or pneumo encephalography A and B normal ventricular system C Ventricles are shifted to the left by a right sided subdural hematoma D The ventricles are enlarged due to brain atrophy following a severe closed head injury

struction of an area of brain and is referred to as porencephaly

Pneumo-encephalogram and Ventriculogram in Surgical Cases

Subdural Hematoma A unilateral subdural hematoma will almost always be revealed in a satisfactory air study The hematoma causes a shift of the ventricular system to the opposite side, just as does any expanding lesion (Fig 10C) It is not necessary to

take roentgen ray films with the patient in the upright position for diagnosis However with the patient upright, occasionally a characteristic air shadow may be present between the inner surface of the clot and the brain near the midline

When the subdural hematoma is bilateral there may be no ventricular shift and the diagnosis can be overlooked. Usually the clot on one side is larger and hence displaces the ventricles

Intracerebral hematomas also can be demonstrated by adequate air studies. A lesion of this type displaces the ventricles to the opposite side and often in an asymmetrical fashion. The resulting distortion of the ventricular system appears much the same as with any other intracerebral mass such as brain tumor or chronic brain abscess.

ANGIOGRAPHY

Carotid angiography or arteriography is useful in the diagnosis of subdural or intracerebral hematoma. This method was first used by Moniz in 1927 It consists of injecting 35 per cent Diodrast (introduced by Gross 1939) into the carotid artery under local or general anesthesia Serial roentgenograms are taken at about half second intervals as the contrast medium circulates through the arteries and then the veins of the brain and surrounding tissues This method is of particular value when the patient is in good general condition The acute and early subacute cases of subdural hematoma, or those patients who are quite drowsy or stuporous, do not stand the procedure at all well nor do some elderly patients with arteriosclerosis

Angiography in Subdural Hematoma. A subdural hematoma in displacing the underlying cerebral hemisphere creates an avascular zone (in the angiogram) throughout the area occupied by the hematoma. In addition the vessels in the compressed brain immediately beneath the blood clot are to some extent obliterated and the deep lying, larger arteries like the middle or anterior cerebral may be pushed toward the opposite side of the head.

Angiography in Intracerebral Hematoma. An intracerebral hematoma displaces and obliterates the Diodrast filled cerebral blood vessels in much the same manner as do certain brain tumors.

THE ELECTRO-ENCEPHALOGRAM ASSOCIATED WITH HEAD TRAUMA[11, 40, 107]

Electro encephalography, discovered by Berger[6] and placed on a sound scientific basis by Adrian and Matthews,[1] is of value in the study of brain injury.

Considerable caution must be used in interpreting electro encephalograms on patients with a history of head trauma. Generalizations concerning the findings in a large group of post traumatic cases should not be applied to the individual patient.

There are many reasons for this caution in interpretation. The EEG findings following trauma are not specific and could have been present before the patient was injured. About 15 per cent of normal people have some abnormality in their EEG. To determine whether an abnormality is the result of a specific injury, repeated EEGs must be taken over a long period. Improvement in the record may take

place or there may be a marked fluctuation in the degree and type of abnormality before the pattern stabilizes. Presumably such a fluctuating abnormality is due to temporary cellular vascular and edematous changes in the cortex subcortex basal ganglia and brain stem

The degree of abnormality of an EEG is judged on the slowness, irregularity and amplitude of the waves the presence of focal abnormalities the relative symmetry in the activity from the two hemispheres and whether or not there is suppression of the normal alpha rhythm

In head injuries with severe clinical manifestations slow waves at four to seven per second are usually present diffusely throughout the record During the first few days after the injury irregular and less often rhythmic delta waves at two to three per second at times of high voltage occur These delta waves are more common in youthful patients With less severe trauma generalized theta waves, either random or rhythmic at four to seven per second and usually of moderate voltage (40 μV) are seen Waves at a frequency of four to seven per second can apparently be caused by traumatic changes in the brain stem

In the mild case of concussion there may be little or no abnormality by one to two days after the accident. Some of these cases have what appears to be a normal record with an alpha frequency at say eight per second. Days to weeks later in some patients the alpha frequency may rise to ten or more per second showing that the previous tracing was abnormal

The amount of abnormality usually lessens with the clinical improvement of the patient but sometimes the reverse occurs, at least temporarily It is

rotation of objects or of the individual, is not very often encountered as a result of trauma When present vertigo is the result of pathology in the vestibular apparatus in the middle ear or its connections in the posterior fossa A form of mild dizziness akin to vertigo, described as a movement of objects or of the patient, but not usually as a violent whirling, is not uncommon These sensations may come on spontaneously or be precipitated by movement of the head or eyes and their origin, like true vertigo, is probably in the middle ear or brain stem

Post-traumatic Nervousness Nervousness occurring with headache and dizziness is quite frequent The patient complains of easy fatigue, that he is irritable, more sensitive to minor annoyances, anxious and often depressed These symptoms are more commonly present in the emotionally unstable, in compensation cases and in the financially insecure, but they are by no means confined to these groups

Treatment of post-traumatic headache, dizziness and nervousness will vary with the degree of injury, type of individual and many other factors In general, as soon as the physical condition permits, these cases seem to do better with a resumption of mental and physical activity and the use of mild sedatives and analgesics for headache Psychiatric care may be required in some Certain localized post-traumatic headaches have been relieved by spinal subarachnoid injection of air in the manner described by Penfield [70]

NEUROSES FOLLOWING HEAD INJURY

It is beyond the scope of this book to describe the neuroses which may be associated with head trauma

It is sufficient to note that a post traumatic neurosis either acute or chronic, may follow a head injury regardless of whether or not organic brain pathology is present. The organic changes however are not the basis of the neurosis

PSYCHOSES FOLLOWING HEAD INJURY

As already discussed organic brain injury may be directly responsible for intellectual impairment and emotional disturbances. To what degree trauma may aggravate an alcoholic, luetic, arteriosclerotic or other organic psychosis depends upon many factors and the writings of psychiatrists should be consulted With regard to any possible relationship between trauma and the functional psychoses such as manic-depressive psychosis or schizophrenia, the reader is referred to the opinion of psychiatrists

8

REHABILITATION

Under this heading consideration is given to early convalescent treatment as well as to later rehabilitation

In the past it was fairly generally accepted that a patient should remain in bed for at least three weeks following a head injury of moderate or severe degree. Opinion is now divided on this point but many authorities agree that such a rule should not be applied indiscriminately Each case should be evaluated separately If an individual is symptom free within a few days after a moderately severe head injury there appears to be no contraindication to allowing him to be up. In fact he will regain his vasomotor and muscular tone more rapidly and will be less likely to have postural dizziness. On this point Penfield[17] quotes Dr K. G McKenzie who has stated "I may say that my practice for some years now has been to allow patients up much earlier than we used to In general my rule now is to let them up a day after the patient feels like getting up for instance, I would not hesitate about letting a compound depressed fracture case get up a day or two after operation if he feels like it."

On the other hand if the patient continues to have symptoms and especially if these have followed a severe injury bed rest should usually be more pro-

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